Anxiety & Depression in Clinical Practice

Edited by : Dr. Mohan Isaac
Dr. Nilesh Shah

Anxiety and Depression in Clinical Practice

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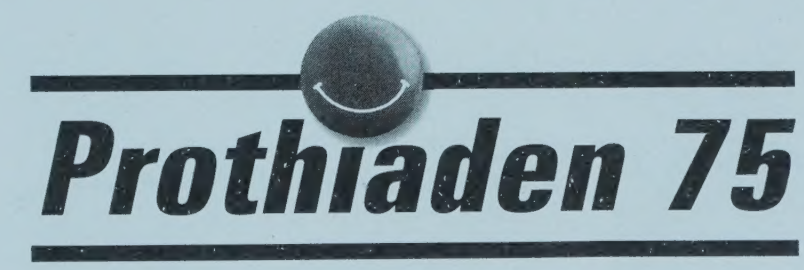

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Anxiety and Depression in Clinical Practice

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Dr. Isaac also holds a number of positions with various non-governmental organizations as well as with the Indian Psychiatric Society. Currently, he is the Vice-President of Medico-Pastoral Association in Bangalore, and the Chairperson of the Psychiatric Education Committee of the Indian Psychiatric Society. He has numerous publications on Community Mental Health and other related topics in National and International journals.

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As a teacher in University of Bombay and Maharashtra University of Health Sciences (MUHS), he has been a guide to a number of medical students pursuing their postgraduate studies in psychiatry. He is also involved in psychiatric training of students pursuing their studies in nursing, social work, clinical psychology, occupational therapy, physiotherapy and special education. He is an active member of Medical Education and Technology Cell (MET-Cell).

He has about 50 publications in various journals and books to his credit and has delivered more than 100 lectures at various scientific meetings. He has participated in number of research projects and is also a member of Ethics Committee of his institute. He has received the best paper awards of Bombay Psychiatric Society, Indian Psychiatric Society (Western Zonal Branch) and Association of Industrial Psychiatry.

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As an administrator he has been instrumental in starting innovative schemes and various facilities for the benefit of the patients.

CONTENTS

PREFACE

It is being increasingly realized that symptoms of anxiety and depression are highly prevalent in general medical population seen in day -to-day clinical practice all over the world. Several authoritative studies have convincingly shown that more than 30% of patients attending primary care clinics as well as specialty clinics in general hospitals have either independent or coexisting anxiety, depressive or other stress related and adjustment disorders. Many symptoms, which patients report are medically unexplained. It is also well known that anxiety and depression are not adequately managed in general health care settings. There are a number of reasons for the neglect of psychological problems in such settings. Due to the profound influence of the Cartesian mind-body dualism, doctors trained in Western medicine are often inadequately equipped to identify and manage symptoms of anxiety and depression in their patients. Patients are also often reluctant to accept psychological explanations of their symptoms for fear of being labeled 'psychiatrically ill'. But, now there is overwhelming evidence to suggest that physical well-being and mental states are intimately connected. Appropriate management of psychological symptoms can improve overall functioning and quality of life.

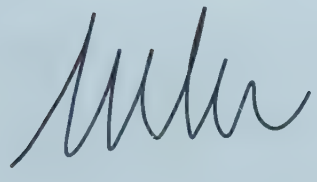
With limited reach of mental health services and grossly inadequate number of mental health professionals, the problem of providing satisfactory mental health care to the population is more severe and acute in India. Therefore, it is very important that general practitioners, family physicians, internists and various other specialists in India equip themselves to identify, diagnose and manage a variety of common psychological symptoms and disorders routinely seen in their every day practice. There is a dearth of clinically useful and practical information on common psychological problems seen in routine clinical practice in India. The main purpose of the book on, 'Anxiety and Depression in Clinical Practice', is to provide family physicians, internists and other specialists with relevant knowledge and information on identification, management (and referral whenever necessary) of common psychological problems.

Efforts have been made to present various symptoms and disorders as they appear in clinical settings in India, in simple, non-technical language without much use of psychiatric 'jargon'. Authors of various chapters consist of leading experts and mental health care practitioners from well-known institutions in different parts of the country who have written from their rich and extensive clinical experience. The topics essentially comprise of three categories. Firstly, there are a number of disorders such as anxiety, panic, phobia, depression, adjustment disorders etc. which are currently referred to as 'common mental disorders'. Anxiety and depression as they appear in three separate populations namely children and adolescents, women and the elderly are dealt within 3 chapters. Lastly several chapters are devoted to symptoms of anxiety and depression in common medical conditions such as stroke, myocardial infarction, various psychosomatic disorders, cancers, sexual dysfunction and in association with major surgery. A chapter discusses insomnia and use of benzodiazepines as these are widespread in general medical practice. Psychological problems related to use of substances such as alcohol, tobacco and other drugs are not dealt with.

While the editors attempted to strike certain uniformity in presentation, the authors had the liberty to write in their own style. Editors' dilemma was to strike a balance between uniformity and variety as well as to reduce overlap, which is unavoidable in a multi-authored book such as this, a task which was interesting but difficult.

We are really grateful to Dr. Roop Majli, a psychiatric colleague, and Dr. Angela Rodrigues and Dr. Manish Garg from Abbott India Ltd., for their help in editing this book. Without their innovative inputs and very useful suggestions it would not have been possible for us to bring out this book in a stipulated time.

It is hoped that this book will add to the ability of doctors to identify and manage symptoms of anxiety and depression in clinical practice thereby improving overall outcomes of their patients.



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Symptoms of Anxiety and Depression in Clinical Practice

Mohan Isaac
Nilesh Shah

Introduction

In clinical practice, a substantial number of patients attending the dispensary of family physicians or the consulting rooms of physicians and surgeons of different specialties have symptoms of anxiety and depression. According to a rough estimate more than 30% of patients having medical or surgical problems have one or more symptoms of anxiety or depression.

It is frequently observed that patients themselves may not be aware about having these symptoms or may not reveal these symptoms spontaneously even if they are distressing.

Occasionally patient's complaints may appear vague or `non-specific' and may not fit into the symptoms known to us. Complaints like *`dimag halka lagata hai', `pet main mitha, mitha, dard rehta hai' `chhati main bharipan mahesus hota hai'* are not uncommon. Many times these are the symptoms of anxiety obscured due to the vocabulary used by the patient to describe them.

Psychosocial stressors and symptoms of anxiety may precede the onset of psychosomatic disorders. An acute attack of bronchial asthma, acute exacerbation of peptic ulcer, sudden rise in blood pressure, an attack of migraine, are frequently precipitated by anxiety provoking situations.

Though development of anxiety symptoms subsequent to the diagnosis of some serious or life threatening illness like cancer may be considered as normal or physiological reaction, these patients and their close relatives do require appropriate intervention and support for the same.

It is a common clinical experience that an anxious patient tends to overreact. He may react to an insignificant abnormality in ECG or X-ray as if he has developed some major life-threatening problem. These type of patients may get preoccupied with their illness and may assume a sick role.

A patient having concomitant symptoms of anxiety or depression may not be able to cooperate and may be non-compliant to the recommended treatment. A depressed patient may not be able to give his full cooperation in physiotherapy sessions for his post-stroke motor deficit.

Perception of pain depends to a great extent on the emotional state of the patient. A depressed or an anxious patient may perceive his mild pain as very severe and unbearable as depression reduces the ability to tolerate pain.

Therefore, in our clinical practice, it becomes mandatory to inquire about symptoms of anxiety and depression in all the patients. It is necessary to note that symptoms of anxiety and symptoms of depression do not fall into two watertight compartments. There may be a fair degree of overlap and patients may have anxiety as well as depressive symptoms or one group of symptoms may be more prominent than the other. One may need lots of patience and special skills for eliciting and interpreting these symptoms. Identification of these symptoms and prescribing appropriate treatment will go a long way in reducing the suffering and enhancing the recovery of the patients having these symptoms.

The common symptoms of anxiety are as follows

Physical symptoms of anxiety	Psychological symptoms of anxiety
• Chest pain, palpitation, tachycardia • Chills, hot flushes, excessive sweating • Dizziness, unsteadiness, light headedness, fainting spells (syncope) • Rapid breathing (hyperventilation), difficulty in breathing, choking sensation • Tremors, trembling or shaking • Nausea, abdominal discomfort (butterflies in stomach), diarrhea • Urinary urgency and frequency • Body aches and pains • Restlessness, fidgetyness • Dilated pupils • Brisk reflexes • Rise in blood pressure, (hypertension)	• Agitation, feeling keyed up or feeling on the edge or tensed • Difficulty in concentrating, mind going blank • Excessive concern, worries, apprehension • Easy irritability • Easy fatigability • Excessive thinking • Fear of losing control or 'going crazy' • Fear of dying • Feelings of unreality (derealization), feelings of being detached from oneself (depersonalization) • Difficulty in falling asleep

The common symptoms of depression are as follows

Physical Symptoms of Depression	Psychological Symptoms of Depression
• Constipation • Dryness of mouth • Decreased appetite, weight Loss • Decreased motor activity • Insomnia or hypersomnia • Easy fatigability and loss of energy,	• Diminished interest or pleasure in activities of daily living • Diminished ability to think, indecisiveness • Diminished desire to socialize • Sadness of mood, crying spells • Intolerance to noise • Decreased sexual drive • Feelings of hopelessness, worthlessness, helplessness • Excessive inappropriate guilt • Recurrent thoughts of suicide or death

How to elicit and interpret the symptoms of anxiety and depression?

As mentioned earlier, patients may not spontaneously reveal these symptoms and therefore it may be necessary to ask the patients specifically about these symptoms.

Ask for the presence of these symptoms especially

- When patient presents with multiple and 'vague' somatic symptoms.
- When patient appears to be tensed or upset.
- When patient starts crying during the interview.
- When patient's reaction seems to be exaggerated.
- When there is a history of some precipitating factors.

One may prefer to start with a non-leading question like `how are you feeling today?' 'How is everything?' or 'How are you doing?' etc.

If the patient does not reveal any symptom and appear to be defensive or uncomfortable about sharing his feelings, one may need to reassure the patient and take him into confidence. You may talk to the patient when his relatives are not around or you may tell the patient that 'whatever he shares with you will be confidential and would not be revealed to anyone without his permission'.

Some patients may not be able to express their own feelings very fluently and you may have to ask them leading questions like `do you feel anxious?' or 'are you worried about something?'

If one feels that symptoms of anxiety or depression are 'understandable' or 'explainable' or `anybody in that situation would feel so'; it does not mean that these symptoms don't need our attention and treatment. For example `he is bound to feel anxious as he has to appear in the exams or interview'.

It is important to assess the severity of these symptoms and find out about the total duration of these symptoms. It is also necessary to evaluate how the person has been coping with these symptoms and is there any impairment in psychological, social and occupational functioning of the patient due to these symptoms. One may also look into the patient's habitual pattern of reaction to stressful situations in the past and whether his reaction, is in keeping with his usual pattern or is different, this time.

When one finds that

- The intensity of the symptoms is out of proportion to the precipitating factors
- The duration is more than what is usually expected
- The symptoms are causing impairment with his day to day functioning and
- This is not the patient's habitual pattern of reaction to stressful situations

One needs to take appropriate measures for the treatment of these symptoms.

Once the symptoms of anxiety and depression, in a given case, are elicited, it is necessary to come to a proper diagnosis, as these symptoms occur in a variety of psychiatric disorders.

Some of the subsequent chapters describe various psychiatric disorders in which these symptoms are usually encountered while the other chapters in this book address the occurrence of these symptoms in medically compromised patients and in special population like children, elderly individuals and women.

There are different diagnostic systems like DSM-IV (Diagnostic and Statistical Manual-IV) or ICD-10 (International Classification of Diseases-10) used by psychiatrist all over the world. Familiarity with these systems may be somewhat useful as these diagnostic terminologies are widely used in clinical practice.

Most of these patients require acute management and maintenance treatment extended over a period of few months. It is well recognized that majority of these patients can be effectively treated by a well-informed family physician. With some experience of using psychotropic drugs and training in counseling, family physicians should be able to treat these patients confidently.

Lack of awareness about psychiatric problems among patients, reluctance to visit a psychiatrist and non-compliance to treatment are some of the common problems encountered in the management of these patients.

For creating the awareness about these disorders, reducing the stigma of having a psychiatric disorder and for appropriate long-term management, a fine cooperation and coordination between psychiatrists and family physicians is of utmost importance.

Generalized Anxiety Disorder

Y.C. Janardhan Reddy

Case of Generalized Anxiety Disorder

A 25 year old married housewife, mother of two girls presented with 3 years history of headache and mental tension. The headache has been present continuously for most part of the 3 years with exacerbations when her tension worsens. She has not been sleeping well and has difficulty in falling asleep with multiple awakenings. She has been worrying excessively about her children's health and their future marriage prospects as both her children are girls. In addition, she worries that her husband who is working in a private factory earning a modest amount just enough to run the family may lose his job. Her husband also has the additional responsibility of getting his younger sister married in to a good family. She constantly worries over these issues, although she is aware that her children are healthy and are still very young to think of their marriage. Her husband has no real prospect of losing his job. In fact, there is a possibility of him getting promotion. Her husband who accompanies her feels that her worries are exaggerated. While she realizes that her worries are excessive, she finds it difficult to control them. She has been feeling tense and edgy for most part of the last 3 years with palpitations, trembling, dryness of mouth and feeling dizzy. When her tension worsens, she has abdominal discomfort and experiences choking sensation. Because of her worries and constant tension, she finds it hard to concentrate on household tasks. She has been finding it difficult to even complete reading the news papers and story books because of poor concentration. There are times, when she gets extremely irritable with her children over trivial matters. Because of constant tension and worry, she is depressed. She believes that there is no end to her problems and on instances has thoughts of dying. Her mood is always low and finds no pleasure in daily activities.

She has consulted neurologists for her headache and cardiologists for her palpitations. She has been extensively investigated with EEG, ECG, CT scan, and many blood tests including thyroid function tests. However, none of the investigations have revealed any abnormalities.

It is evident from her history, that the main symptoms are pervasive anxiety and worry over issues related to children, husband's job and sister-in-law's marriage. Both the patient and her spouse admit that her worries are exaggerated. Her anxiety is associated with tension type of headache and autonomic hyperarousal. Since her focus of worry is family circumstances and anxiety is pervasive she gets a diagnosis of Generalized Anxiety Disorder (GAD). She also has co-existing depression. Her anxiety symptoms are not episodic and do not occur in specific circumstances and situations. Therefore, she has no symptoms to suggest panic disorder or phobias. Her investigations, many of them unnecessary, have not revealed evidence of any medical conditions that can explain anxiety.

This patient would benefit from the use of antidepressants since she has both GAD and depression. Benzodiazepines are not preferred drugs since they have no antidepressant property. Moreover, use of benzodiazepines is tricky in her case as she may have to be on the medicines for a prolonged period and that might result in dependence and subsequent problems in withdrawal. Brief education about her illness and cause of her somatic symptoms is mandatory since she has been extensively investigated for physical illnesses. It would also help her understand the relationship between her perception of stress, and anxiety.

Generalized Anxiety Disorder

Generalized anxiety disorder (GAD), earlier called anxiety neurosis is a disabling and chronic condition that is common in general practice and in many medical settings. However, it is often unrecognized and under diagnosed in medical settings because of many physical symptoms that accompany anxiety. Associated physical symptoms, mainly symptoms of autonomic hyperarousal often lead physicians to search for physical illnesses with resultant delay in treatment. The patients who suffer from GAD frequently have co-existing depression.

Epidemiology

Life-time prevalence rates of GAD vary across studies. The recent studies report prevalence rates of about 5% in the general population (Kessler et al., 1994). However, the rates of GAD are likely to be much higher in the clinic populations of primary health care system and general medical settings. The GAD is about twice as common in women as in men.

Etiology

Both biological and psychosocial factors seem to play a role in the genesis of GAD (Brouette and Goddard 2002). Genetic factors appear to predispose to development of GAD in some individuals, but genetic factors do not yet account for all cases. Several neurotransmitter systems (catecholamines, serotonin, GABA and cholesystokinin) have been implicated in the aetiology of GAD. Patients with GAD also have hyperactive hypothalamo-pituitary-adrenal axis but thyroid axis appears unaffected. They also have shorter interbeat variability on ECG, which may be the result of an inflexible response to stress and a requirement for a prolonged time to recover from a stressor. There is also some evidence to show that amygdala plays a central role in the mediation of fear reactions.

There is an association between stressful life events and GAD. The experience of even one very important unexpected negative life event is associated with a threefold increase in GAD. A variety of stressors have been associated with increased risk of GAD, including early parental death, rape or combat and chronically unhappy marital and family relationships. Parenting also seems to play a role in the development of GAD. A parental style characterized by overprotection, criticality and intrusiveness combined with a lack of warmth and responsiveness toward the child could contribute to anxiety.

Clinical features

The symptoms of GAD are numerous and highly variable (WHO, 1992; Papp and Kleber 2002). Restlessness, easy fatiguability, poor concentration, irritability, muscular tension, difficulty to relax and sleep disturbances are some of the common symptoms. Symptoms of autonomic hyperarousal are frequently the presenting problems. These include palpitations, tremulousness, sweating, sensations of shortness of breath, feeling of choking, chest discomfort, feeling dizzy and abdominal discomfort. The autonomic symptoms are usually prominent at the height of anxiety. The motor tension usually results in frequent headaches (tension type headache) and chronic muscle pain in the shoulder, neck and lower back.

While the above mentioned symptoms are common in GAD, the characteristic feature is excessive anxiety and worry (apprehensive expectation of negative outcomes) about various events and activities such as concerns about family and interpersonal relationships, work, school, finances, and health. The GAD patients most characteristically worry excessively over minor matters such as daily hassles and time management. The person suffering form GAD finds it difficult to control the worry. The anxiety and worry is usually present for most part of the day and patients typically report that they have been suffering from worry and anxiety for many years. A diagnosis of GAD is usually made when symptoms persist for at least several months. A negative answer to the question 'Do you excessively worry about minor matters?' often rules out a diagnosis of GAD.

Differential diagnosis

Depression

The most difficult differential diagnosis is between chronic depression (dysthymia) and anxiety. This is because both the disorders share many features and often co-exist in the same patient. Both the disorders have insidious onset and protracted course. Symptoms such as poor concentration, fatigue, easy irritability and sleep disturbances are common to both the disorders. Sometimes, symptoms of anxiety represent the prodrome to major depression or panic disorder. However, severe depressive symptoms such as suicidal ideation, feelings of worthlessness, hopelessness, helplessness and guilt are more characteristic of depression, whereas excessive vigilance, scanning, somatic symptoms indicate anxiety disorder. However, it is well known that significant proportion of GAD patients also suffer from depression and in such patients dual diagnoses are made. Identification of depression in GAD is very important because the pharmacological approach could vary in those with co-existing depression from those without.

Panic disorder

The most distinguishing feature of this disorder is the attacks of anxiety or panic. In GAD, onset is typically insidious and anxiety is usually present for most part of the day. In panic disorder, the anxiety attacks are brief and occur in episodes. Panic attacks typically begin spontaneously and later tend to occur in specific situations. They are typically associated with severe autonomic symptoms and thoughts of catastrophic consequences (e.g., losing control, having a heart attack or 'going crazy'). Sometimes, it is difficult to differentiate GAD from panic disorder, because both co-exist and share similar symptoms. Moreover, anticipatory anxiety characteristic of panic disorder i.e., fear of having additional panic attacks is often mistaken for free-floating anxiety of GAD.

Social phobia

Those with social phobia experience anxiety only in social situations. For example, they experience anxiety when they have to perform or speak in front of others (public speaking), talk to superiors and members of opposite sex, participate in small groups, attend social gatherings (weddings, birth day celebrations) or have to write and eat in front of others. There is extensive avoidance of these social situations and the sufferers harbour beliefs that they may behave in an embarrassing way inviting ridicule from others or that they may be criticized or negatively evaluated. In GAD, anxiety is not confined to social situations and is usually about daily activities and events which are often trivial in nature.

General medical conditions

Following common general medical conditions can cause symptoms and signs that resemble GAD (Spiegel and Barlow 2000).

- Cardiac conditions (arrhythmias, coronary insufficiency, heart failure)
- Endocrine conditions (hyperthyroidism, hypoparathyroidism, hypoglycemia).
- Neurological disorders (temporal lobe epilepsy, vestibular nerve disease).
- Respiratory conditions (asthma, obstructive lung disease, pulmonary embolism).

Certain medications cause symptoms and signs that resemble GAD. Use of psychotropics (sedative-hypnotic and antidepressant withdrawal), respiratory (bronchodilators, beta adrenergic stimulants) and cardiovascular drugs (antihypertensives, antiarrhythmics), thyroid hormone, non-steroid anti-inflammatory drugs and anti-cancer drugs can cause anxiety-like symptoms. In addition, withdrawal from alcohol and other drugs of abuse can also produce GAD like picture.

It is important to take detailed medical and drug history to rule out these conditions that can mimic GAD. However, 'pathological worry' and chronic nature of the illness characteristic of GAD help in differentiating it from other medical conditions.

Co-existing psychiatric disorders

GAD usually occurs with other anxiety and mood disorders. Most common among these are depression, simple and social phobias, and panic disorder (Kessler et al., 1994; Wittchen et al., 1994). Personality disorders are also common in patients with GAD.

Course and Prognosis

The onset is usually in the 20s, but it is not unusual to find GAD in children and elderly. In fact, many GAD patients report life-long tendency to excessive worry and anxiety over trivial matters. The illness tends to run a chronic course with waxing and waning symptoms.

Management

Pharmacotherapy

The drugs effective in the treatment of GAD include benzodiazepines (alprazolam, clonazepam and diazepam), 5-HT1A receptor agonists (buspirone), tricyclic antidepressants (imipramine, amitriptyline, clomipramine, dothiepin), selective serotonin reuptake inhibitors (SSRIs), and serotonin-noradrenaline reuptake inhibitors (SNRIs) (Spiegel and Barlow, 2002; Sussman and Stein, 2002).

Benodiazepines

The benzodiazepines are effective anxiolytics with rapid onset of action and are well tolerated by patients. However, considering the chronic nature of the illness, and therefore the need to use drugs for longer periods, there are some concerns about using benzodiazepines as the first line drugs.

Although they are associated with abuse potential, more worrisome is the dependence, even with discontinuation of relatively low doses. They are shown to affect sustained remission of anxiety symptoms without dose escalation over 6 months and longer. After acute use, symptoms of relapse and/or rebound anxiety (return of anxiety to a level above the pretreatment baseline) frequently occur.

Given the potential difficulties involved in withdrawing benzodiazepines, many clinicians would argue that they should not be used as first-line agents in the treatment of GAD. However, for some patients benzodiazepines may be the preferred drugs in view of their rapid onset of action and better tolerability. More over, they are useful agents when patients do not tolerate other drugs or show poor response to them. In such cases, the symptoms of anxiety may be reduced without any tolerance or dose escalation. At the time of withdrawal, gradual tapering regimes or switching to a benzodiazepine with a longer half-life (e.g., clonazepam, diazepam) may have to be employed. Some clinicians use benzodiazepines along with other drugs in the first few weeks because of their rapid onset of action and thereafter gradually withdraw them to avoid dependence.

Generally, benzodiazepines with longer half-lives (clonazepam, diazepam) are preferred, which can be taken once or twice daily. A typical starting dose of diazepam is 5-10 mg/day which is advanced every few days to a maximum of about 30-40 mg/day. Similarly, clonazepam is started at a dose of 0.5 mg twice a day and later increased at weekly intervals to a maximum of 2-3 mg/day. Dose titration is made gradually (weekly) depending on the clinical response and tolerability. Most patients experience significant improvement in the first 1 or 2 weeks of treatment.

Buspirone

This drug has no abuse potential and does not cause dependence. However, its efficacy is not as well established as benzodiazepines. Some trials have shown buspirone to be comparable in efficacy to benzidiazepines at doses of 20-40 mg, but there are few negative trials too. Moreover, buspirone has a slower onset of action than benzodiazepines. Buspirone is an useful option in patients who do not demand immediate relief of symptoms and in those who do not have co-existing severe depression. Its favorable side-effect profile is particularly attractive.

The typical starting dose of buspirone is 15 mg/day in divided doses, which is increased by 5 mg/day every few days to a target dose of 30 mg/day. If the response is insufficient after 3 to 4 weeks of treatment with that dose, it may be gradually hiked up to a maximum of 60 mg/day.

Antidepressants

This class of drugs includes tricyclics, SSRIs and SNRIs. The most commonly used tricyclic antidepressants are imipramine, amitriptyiline, and clomipramine. Most tricyclics have anxiolytic property in addition to their antidepressant property. The SSRIs (fluoxetine, sertraline, paroxetine, citalopram and fluvoxamine) are being used increasingly in the treatment of GAD although adequate data supporting the use of most SSRIs for GAD is not yet available. The selective serotonin reuptake inhibitor (SSRI) paroxetine has been approved recently for the short-term treatment of GAD. The SNRI, venlafaxine has also been recently approved for the treatment of GAD. The antidepressants in general have a slower onset of action (3-4 weeks) but have no abuse potential and are easier to

withdraw than the benzodiazepines. However, tricyclics are associated with sedation and troublesome anticholinergic side effects (e.g., dryness of mouth, constipation) that often contribute to treatment discontinuation. Therefore, dose titration has to be done gradually. The SSRIs on the other hand, are associated with lower incidence of anticholinergic side effects and sedation but are known to cause more sexual side effects. Venlafaxine is a well tolerated drug, but it may cause hypertension in higher doses. Therefore, venlafaxine may have to be used cautiously in those who already have hypertension.

Tricyclic antidepressants are usually given in the range of 75 to 150 mg/day. Patients may respond to the lower range of dose and therefore, dose hike is generally recommended after 3-4 weeks of treatment. Venlafaxine may be effective at a lower dose of 75 mg/ day. However, if the response is inadequate, dose may be increased by 75 mg/day every 1-2 weeks to a maximum of 225 mg/day. Venlafaxine has a short half-life. It is advisable to use extended-release form that can be administered once a day to improve compliance. The SSRIs are used in the following doses: fluoxetine 20-40 mg/day; sertraline 100-150 mg/day; paroxetine 20-40 mg/day; citalopram 40-60 mg/day; and fluvoxamine 200-300 mg/day.

To summarize, there are several effective agents to treat GAD but many medications have unfavorable side effects. The long-term use of benzodiazepines in GAD is controversial because of problems associated with withdrawal. However, benzodiazepines could be used judiciously in the acute treatment of GAD because of its rapid onset of action. There is little consensus on algorithm for pharmacotherapy of GAD. Nevertheless, given the high degree of comorbidity with depression and panic disorder, there are compelling reasons to prefer antidepressants to benzodiazepines over the long haul. Although, the onset of action is delayed they, have a broad spectrum of action, are easier to discontinue, and are not subject to misuse. Some patients with GAD may have comorbid obsessive compulsive disorder (OCD) and social phobia. In such cases, SSRIs are preferred over tricyclics because they are effective in treating OCD and social phobia as well. For patients who are intolerant of these agents, psychotherapy is an equally effective alternative option. Where feasible, psychotherapy could be the first treatment option and can be employed in combination with the drugs.

Psychosocial intervention

Psychotherapy plays an important role in the treatment of GAD. Several techniques are employed to treat GAD. These include psychoeducation, cognitive therapy (restructuring of negative thoughts related to perception of danger and threat and its consequences) and relaxation exercises. In general practice, it may be difficult to practice cognitive therapy because it requires special training and is time consuming. However, the other two techniques can be employed with relative ease. Psychoeducation about the illness is an important aspect of treatment. Many patients who have come in for treatment are not told about their diagnosis with resultant misconceptions about symptoms. It gives a great sense of relief for patients to know that their somatic (physical) symptoms are not the result of physical illnesses and that they are the result of excessive worry and anxiety. Simple relaxation techniques such as progressive muscle relaxation techniques, yoga, transcendental or other types of meditation may be helpful for the relief of anxiety.

Panic Disorder

Ajit Avasthi
Paramjeet Singh Khurana

'A case of recurrent heart attacks'

Mrs. Surjit, a 35 year old housewife, was referred with a history of what she described as getting 'repeated heart attacks'. She recalled that her problem began 10 years back, when she had delivered her only child. The first attack occurred while she was working in the kitchen, when she suddenly felt that, there was a dramatic increase in her heartbeat. She also felt an intense stabbing pain in her chest and had difficulty in breathing. She started sweating and trembling, felt dizzy, had tingling sensations in her left arm and feared that she was going to die from a heart attack. She immediately rushed to a nearby physician. An electrocardiogram was performed immediately and was reported to be normal. Since then, Mrs. Surjit has complained of occurrence of similar attacks, with each attack lasting for a period of 15-30 minutes. The frequency of these attacks, have been four episodes per month. During these attacks, she calls for help and frequently seeks medical advice. Over the past 10 years, she has had 'far too many', medical investigations, each of them reconfirming and reassuring her that she has no cardiac disorder. After her first few attacks, she developed a fear of having an attack in situations, where she will be away from home or when she will be present at places where medical help will not be easily available. Since then she avoids crowded places such as banks, marriage parties, and cinema-houses, where quick escape might be blocked. The attacks still occur and are observed more frequently in those situations where she fears them the most. She recognizes that both her symptoms and her avoidance behavior are unreasonable and excessive, but all the same they dominate her life. She feels mildly depressed and restless and has difficulty falling asleep. Her self-confidence is low, and she also has difficulty in concentrating.

Initially Mrs. Surjit was treated with a variety of beta-blockers for an 'irritable heart'. Her family physician then additionally prescribed diazepam, which she has consumed in a dosage of 5 mg three times a day for the past 8 years, but without any significant improvement.

The patient described herself as a 'nervous type' who often felt tense and apprehensive in unusual situations. She has always been self-conscious, sensitive to criticism, and reluctant to be involved with other people unless she knew them well. Her self-confidence has always been low, and she has a tendency to feel inferior to other people. She exhibits a tendency to manifest, depressive reactions when faced with disappointments or criticism.

On examination, Mrs. Surjit did not appear depressed but was tense and spoke quickly, with a sense of urgency. She described her complaints spontaneously and vividly. She appeared to be quite intelligent. No psychotic symptoms were detected. Physical and neurological examination revealed no physical disorder. Electrocardiographic, electroencephalographic, and serological examinations were all normal and even the thyroid parameters were within the normal range.

Mrs. Surjit, is suffering from panic disorder as she is getting recurrent discrete episodes of panic associated with intense fear and discomfort. These episodes were sudden in onset, would last for about 15-30 minutes and recurred at a frequency of about 4 per month. She is suffering from this disorder for last 10 years. Apart from the fear of dying from a heart attack the panic attacks also includes palpitations, sweating, trembling, chest pain, breathing difficulties, dizziness, and tingling sensations.

Panic disorder

Panic disorder draws its name from the Greek god Pan, god of flocks. Pan was known for suddenly frightening animals and humans out of the blue. The spontaneous 'out of the blue' character of panic attacks is the principal identifying characteristic of panic disorder and central to its recognition and diagnosis. In the past, these symptoms have been variously referred to as irritable heart, Da Costa's syndrome, neurocirculatory asthenia, disorderly action of the heart, and effort syndrome.

Epidemiology

Epidemiological data obtained from different countries have documented similarities in lifetime prevalence (1.6%-2.2%), age at first onset (20s), higher risk in females (about two fold), and symptom patterns of panic disorder (Weissman, 1997). While the full-blown syndrome is usually not present until early adulthood, limited symptoms often occur much earlier (Moreau, 1992). Several investigators have documented cases of panic disorder prepubertally. One-third to one-half of individuals diagnosed with panic disorder in community samples also have agoraphobia (Weissman, 1997). Among individuals with panic disorder the lifetime prevalence of major depression is 50%-70% (Lesser, 1989). For individuals with both panic disorder and depression, the onset of depression precedes the onset of panic disorder in one-third of this population, while the onset of depression coincides with or follows the onset of panic disorder in the remaining two-thirds. Approximately one-third of patients with panic disorder are depressed when they present for treatment (Lesser, 1989). In the Epidemiologic Catchment Area (ECA) study, subjects with panic symptoms or disorders, as compared to other disorders, were the most frequent users of emergency medical services and were more likely to be hospitalized for physical problems (Klerman, 1991). Patients with panic disorder, especially with comorbid depression, were at higher risk for suicide attempts (Weissman, 1989), impaired social and marital functioning, use of psychoactive drugs, and substance abuse (Markowitz, 1989).

Etiology

Genetic predisposition

Family studies using direct interviews of relatives and family history studies have shown that panic disorder is highly familial. Overall, evidence from family and twin studies suggests that panic disorder involves modest inheritability of around 30 to 40 percent.

Role of neurotransmitters

Various neurotransmitters like norepinephrine, serotonin and GABA have been implicated in the etiology of panic disorder.

Precipitating events

Precipitating events have been reported in 60-96% of cases. These have often centered on separation or loss, relationship difficulties, taking on new responsibility, and physiological stressors (e.g. childbirth, surgery, hyperthyroidism). There are many studies suggesting that traumatic early events like parental separation, sexual abuse may increase the vulnerability to panic disorder.

Clinical features

- The attack may start suddenly with palpitation, chest pain, sweating difficulty in breathing, choking sensation and feelings of impending doom.
- There may be associated flushing, giddiness, abdominal discomfort and goose flash.
- The typical attack may last for about 15 to 20 minutes.
- The frequency of such attacks may vary from once in 2-3 weeks to 2- 3 attacks or more in a week.
- After repeated attack of panic, individual may develop anticipatory anxiety and may start avoiding certain situations associated with panic.
- He may also avoid situations in which getting help or escape is difficult if he gets a panic attack; e.g. he may avoid staying alone or going out of house. He may not go to overcrowded places like market or close place like cinema hall and may avoid traveling by crowded bus or train. This avoidance behavior is known as 'agoraphobia'.
- In severe cases, it may be very disabling and may make a person practically housebound.

Differential diagnosis

Psychiatric conditions: Panic disorder must be distinguished from other conditions that have panic symptoms as associated features

- **Depression:** Panic disorder and depression may often occur together. Proper recognition of comorbid depression is especially important because of the marked increase (four fold) in suicide attempts in these patients.

- **Generalized anxiety disorder (GAD):** The difference between panic disorder and generalized anxiety disorder (GAD) depends on whether patients have panic attacks or whether they have multiple, unrealistic and excessive worries about most aspects of life, not just panic attacks. These worries in GAD often concern money, health, children, work problems, etc.

- **Social Phobia:** Anxiety Symptoms in Social phobia centre not around the occurrence of panic attacks, but are confined entirely to social situations where the individual fears embarrassment and humiliation.

- **Specific phobias:** Panic attacks in social phobia occur in very specific situations, e.g., acrophobia (fear of heights) or in the presence of specific objects, e.g. zoophobia (fear of animals, snakes) etc.

- **Posttraumatic stress disorder (PTSD):** patient may have many panic-like symptoms, but their illness begins quite specifically after a traumatic experience 'outside the range of usual human experience' where they feel threatened with death or serious injury. The traumatic event is persistently re-experienced in the form of distressing recollection of events or recurrent dreams.

- **Obsessive-compulsive disorder (OCD):** In patients suffering from OCD the panic attacks are usually associated with their obsessional concerns, e.g. obsessions about contamination.

Medical conditions: Panic like symptoms do occur in various medical conditions as mentioned in table

Medical conditions that produce panic like symptoms
I. Cardiovascular Disorders Angina, hypertension, hyperactive α-adrenergic state, mitral valve prolapse, myocardial infarction, paroxysmal a trial tachycardia
II. Neurological Disorders Cerebrovascular diseases, epilepsy, Huntington's disease, infections, Meniere's disease, migraine, multiple sclerosis, transient ischemic attack, tumors, Wilson's disease, temporal arteritis
III. Endocrine Disorders Addison's disease, carcinoid syndrome, Cushing's syndrome, diabetes, hyperthyroidism, hypoglycemia, hypoparathyroidism, menopausal disorders, pheochromocytoma, premenstrual syndrome
IV. Drug Intoxication Amphetamines, amyl nitrite, anticholinergics, cocaine, hallucinogens, marijuana, nicotine, theophylline
V. Drug Withdrawal Alcohol, antihypertensives, opioids, sedative-hypnotics
VI. Other Conditions Asthma, pulmonary embolus, anemia, CRF, anaphylaxis, B_{12} deficiency, electrolyte disturbance, heavy metal poisoning, systemic infections, systemic lupus erythematosus, uremia.

Course and Prognosis

Panic disorder usually has its onset during the late adolescence or early childhood. It is generally a chronic disorder, with 30-40% of patients seeming to be symptom free at long term follow-up; about 50% having symptoms that are mild enough not to affect their lives significantly, and about 10-20% continuing to have significant symptoms. Alcohol and other substance dependence occur in about 20-40% of all patients and obsessive compulsive disorder may also develop. Patients with good premorbid functioning and a brief duration of symptoms tend to have good prognosis (Roy-Byrne, 1995).

Management

The specific components of management include performing a diagnostic evaluation; establishing and maintaining a therapeutic alliance; monitoring the patient's psychiatric status; providing education to the patient and, when appropriate, to the family about panic disorder; enhancing treatment compliance; and working with the patient to address early signs of relapse (Gorman, 1998).

Patients with comorbid psychiatric problems (especially with suicidal tendencies), substance abuse problems, significantly impaired functioning caused by avoidant behavior, or poor response to therapy should be referred to a psychiatrist.

Pharmacotherapy

Pharmacotherapy for panic disorder includes use of certain antidepressants (especially Selective Serotonin Reuptake Inhibitors (SSRI)) or a high-potency benzodiazepine. Only paroxetine, sertraline and alprazolam are currently approved by the US Food and Drug Administration (FDA) for panic disorder. Benefits of long-term drug treatment are controversial but long-term therapy does appear to reduce the risk of relapse. Physicians must evaluate the dependence liability and abuse potential of benzodiazepines when considering long-term treatment (Nemeroff, 1999).

Antidepressants

SSRIs are first-line agents because of their documented efficacy, safety and tolerability profile. Tricyclic antidepressants and SSRIs, particularly fluoxetine, can cause a 'jitteriness' syndrome (also called activation) that presents as anxiety, insomnia, agitation, and diarrhea early in the treatment course. To minimize this syndrome, which may reduce patient's compliance with the regimen, one half to one third of the usual starting dose (e.g., paroxetine 10 mg, fluoxetine 10 mg) may be used and increased slowly over 3 or 4 weeks until a response is observed.

Concurrent use of a low-dose benzodiazepine also may alleviate these symptoms while the SSRI is being titrated. Long-term SSRI use can be associated with sexual dysfunctions in both men and women. This concern should be discussed with the patient. With the SSRIs, a clinical response may not be noted before 3-6 weeks. If a rapid response is needed for severe cases, benzodiazepine therapy may be preferred. Emerging data with venlafaxine and nefazodone are promising for treatment of panic disorder (Gorman, 1998).

Benzodiazepines

The SSRIs have largely replaced the benzodiazepines as first-line treatment of panic disorder. High-potency benzodiazepines (e.g., alprazolam, clonazepam, lorazepam) are effective at rapidly relieving anxiety. Low doses should be used initially (e.g., alprazolam 0.5 mg two or three times daily with subsequent dose escalation based on the clinical response and the tolerability profile. High doses are sometimes needed (e.g., alprazolam up to 10 mg/day). However, side effects may limit the ability to increase the dose to obtain maximal benefit. Some patients are unwilling to take benzodiazepines because of dependence or tolerance issues. The primary side effect of benzodiazepine therapy is sedation, which can be reduced by lowering the dose. Tolerance to this effect may develop over a period of time (Gorman, 1998).

Once effective, pharmacotherapy should generally be continued for 8 to 12 months. Abrupt discontinuation of treatment may lead to relapse in substantial number of patients (Ballenger, 2000).

Psychological treatment

Cognitive therapy

Cognitive therapy helps patients identify and change misinterpretations of bodily sensations and substitute them with more realistic interpretations (e.g. explaining to patients that panic attacks are time limited and not life threatening). Behavioral procedures include inducing feared situations (by hyperventilation), focusing attention on the body, or reading words representing the feared situations, in order to demonstrate possible causes of patient's symptoms, and stopping safety behaviors (such as holding onto solid objects when feeling dizzy), in order to help patients disconfirm their negative predictions about consequences of their symptoms (Clark, 1994).

Relaxation therapy

Applied relaxation techniques, like that of Herbert Benson's relaxation training and the one devised by Ost are commonly used. In Ost's procedure, the patients are taught to identify the early signs of panic and relax rapidly. As panic attacks can occur in any situation, the training consists of a series of stages, in which patients are taught to relax more and more quickly while performing the everyday activities, such as walking and shopping. Home work assignments include twice-daily relaxation practice (Ost ,1991).

One direct approach to control panic attacks is to train patients about how to control the urge to hyperventilate by respiratory training, since hyperventilation associated with panic attacks may cause dizziness and faintness.

Psychosocial therapies, such as family therapy and insight oriented psychotherapy, can also be of benefit in the treatment of panic disorder.

Combined psychological and pharmacotherapy are more effective than either therapy alone. Cognitive therapy, administered in parallel with alprazolam maintenance and taper, is effective in preventing relapse after drug discontinuation. In a comprehensive treatment program, initial pharmacotherapy should use alprazolam for its quick onset, with slow titration of antidepressants, and then tapering of alprazolam, with application of cognitive behavior therapy to prevent relapse.

Conclusion

Panic disorder is a chronic but treatable problem, associated with a high degree of social and work impairment, poor quality of life, and frequent relapses when drug treatment or psychotherapy is withdrawn. Often unrecognized, it is associated with excessive use of medical services (especially emergency room and primary care visits). This can have a chronic course and can be associated with significant morbidity. The care of patients with panic disorder involves a comprehensive array of approaches that are designed to reduce the frequency and severity of panic episodes, reduce morbidity, and improve patient functioning.

Depressive Disorders

Manoj Bhatawdekar

'Could his life have been saved?'

Mr. Dilip Sathe, a 55 year old Marathi-speaking, retired bank employee from Mumbai was reported to have died in a 'railway accident'. He was run over by a moving train and his body was found on the railway tracks .He apparently had the 'habit' of crossing the railway tracks instead of using the bridge, as reported by his friends. The friends guessed that he went wrong in his 'judgment' while crossing the tracks on that day and met with an 'accident'. A doctor friend of Sathe, however, spoke to his wife and elicited some information regarding Sathe's behaviour in the past six months. Sathe had opted for voluntary retirement last year. He was not interested in taking up another job. He would spend his time reading, watching television and helping his wife in household work instead. He seemed to enjoy this lifestyle for the first six months. However, since the past few months his wife had noticed a considerable change in his behaviour. He seemed worried and tense most of the time for no apparent reason.

But he would deny this whenever questioned. He would wake up at about 4 am. and then would find it difficult to get sleep. He tried taking some sleeping pills on his own but in vain. He would feel excessively tired throughout the day. He lost his appetite and his intake of food dropped to almost fifty percent of usual. As a result of this he lost 8 kg over a period of 3 months. He also complained of constipation very often. He stopped going for his morning walks, stopped watching TV, and stopped reading books. He attributed this to 'not feeling like' doing any of these activities. He would often remark that he had wasted all his life so far. He would feel guilty that he had not saved enough money for his children and feared that they would never forgive him for that. Whenever his wife tried to talk him out of this he would only be temporarily be convinced, reverting soon to his previous thoughts. He gradually started feeling more and more helpless about his lack of control over his life. He could not figure out any reason in his environment for what he was going through. This made him think that he was unnecessarily creating problems for himself and his family. He would ruminate over this thought and would conclude that life was not worth living. His friends who visited his house used to offer different kinds of advice to him, such as doing yoga, going for a walk in the morning, taking Reiki, doing meditation, engaging in spiritual reading etc., none of which Mr. Sathe found worth considering. His wife's idea of giving him psychiatric treatment was almost ridiculed by the friends since they believed that it would be a lifelong crippling treatment.

Finally, the mishap took place. His wife had no doubt that he had committed suicide. 'Could his life have been saved?' asked his wife to the doctor friend. One can easily understand how difficult it must have been for the friend to answer this question.

Major Depression

Mood disorders constitute disorders, which have a persistent disturbance in mood as the prominent feature. Major depression is one such disorder that hampers the quality of life of an individual remarkably. This is also a disorder that can lead to life-threatening complications such as suicide. It is therefore important for a clinician to recognize the presence of this condition in a case and treat it effectively. Generally, the outcome of treatment is good if the condition is detected early and treated adequately.

Epidemiology

There have been various studies (Kessler et al, 1998) among different patient populations measuring the risk of major depression. Generally, the results all over the world are more or less similar. The lifetime prevalence of major depression is 15.3%, whereas 30 day prevalence is 5.8%. About 73.9% of patients with major depression report recurrent episodes. In these patients role impairment is significant, including suicidal attempts among 21.9% of them. In general, the risk of developing major depression is higher in women than men. About 18 to 23% of all women and 8 to 11% of all men have depressive episodes at some time. 6% of those women and 3% of those men require hospitalization at some time.

Etiology

Genetic factors

There is an increased risk of developing the disorder among blood relatives of patients with Major depression. Twin studies have shown significantly greater risk in monozygotic twins than in dizygotic twins. It is the tendency to have the disorder that is inherited than the actual disorder.

Biological factors

Major Depression was called 'endogenous depression' earlier. This clearly indicates the role of biological factors in the etiology of the disorder. Various disturbances in the circadian rhythms have been noticed in patients with this disorder. A very important factor that is implicated in the aetiology is abnormalities in the neurotransmitter systems. There is a dysregulation in the noradrenergic, serotonergic and at times in the dopaminergic neurotransmitter systems, which primarily produce the disturbance in the mood. At times, some medical conditions such as viral infections can precipitate the condition. Physiological stresses like childbirth, surgery can also be the starting point of the clinical manifestations.

Psychosocial factors

Specific factors such as some losses (e.g. monetary loss, loss of a person etc.) may trigger the onset of the condition. Other stresses such as difficulties with interpersonal relationships, retirement or any other life events may be responsible in the etiology.

Clinical features

The following clinical features are commonly encountered in a depressed patient:

1. Sadness of mood is the most prominent feature. The sadness may be described by the patient as feeling 'down in the dumps', or feeling restless, or feeling 'numb in the head', or as having no feelings etc. Some of them report having physical pains and aches. Some may report feelings of agitation or anger while some may report feelings of hopelessness.

2. The person loses interest in all or almost all pleasurable activities including hobbies and sex.

3. There is a significant loss of appetite resulting in loss of weight; approximately 5% of body weight in a month. The person may feel that he or she has to force himself or herself to eat. Some individuals may have an increased appetite resulting in weight gain.

4. Sleep disturbances are very common in major depression. The commonest is insomnia in the early morning. The person wakes up unusually early and experiences severe restlessness being unable to fall asleep again. At times, the person may sleep excessively.

5. There is either psychomotor retardation or agitation. A person with psychomotor retardation is observed to have slow speech, thinking and movements and becomes less communicative and even mute. A person with psychomotor agitation is unable to sit still, shows wringing movements of hands, paces up and down, or may pull or rub clothes. These disturbances are severe enough to be observed by others.

6. Tiredness, fatigue and lack of energy are common even without much physical exertion. Everyday tasks such as dressing, washing require longer time and appear exhausting.

7. The person feels inappropriately guilty for his or her own actions. Minor difficulties may be interpreted as major failures. There is lack of self-esteem and a tendency to blame oneself owing to the inability to carry out responsibilities. The sense of worthlessness or guilt may be of delusional proportions i.e. the person firmly believes that he or she is guilty and is not amenable to any amount of reasoning.

8. There is a decreased ability to think or concentrate or indecisiveness or forgetfulness. The person's socio-occupational performance deteriorates as a result of these symptoms.

9. Recurrent thoughts of death are very common. The person may wish natural death or may contemplate suicide. There may be just thoughts about ending one's life or the person may make specific plans about committing suicide. This particular symptom is alarming and should never be taken lightly and should always be treated on an emergency basis. It is important to ask about this symptom if the person does not come out with this history. It is a myth that asking about suicide might actually instill the idea in the patient's mind. On the contrary, it motivates the patient to share thoughts about something he or she is embarrassed to talk about.

The above-mentioned symptoms may run a chronic protracted course for years and with a less severity in some individuals. It is then called Dysthymic Disorder or Minor Depression.

The diagnosis of depression is made after a careful history, mental status examination and physical examination. Certain psychological tests can be used to confirm the diagnosis. Psychological rating scales may be used to assess the severity of depression.

Diffrential diagnosis

Drugs such as reserpine, corticosteroids, methyldopa, levodopa, cycloserine, ethionamide, oral contraceptives, and amphetamines can cause depression. Endocrine disorders such as hypothyroidism, Cushing's syndrome, Addison's disease, hyperparathyroidism, infections such as influenza, infectious mononucleosis, and viral hepatitis are associated with depression. Certain

cancers such as carcinoma of the pancreas, tumours of the brain, autoimmune disorders like SLE, neurological disorders like Parkinsonism, stroke and dementias can cause depressive symptoms. Anaemia is very often associated with depression.

Psychiatric disorders

Depressive disorders have to be differentiated from anxiety disorders, schizophrenia and alcoholism.

Course and Prognosis

Most of the patients suffering from major depression recover completely with proper treatment. About 20% of them may be resistant to treatment and may require a combination of various approaches. Patients with dysthymic disorder require drug treatment over a long period of time, along with psychotherapy. Patients with major depression should be observed for a recurrence of symptoms. In some patients symptoms of mania develop, in which case the diagnosis become bipolar disorder. In general, depressive disorders have an overall good prognosis as compared to major psychiatric conditions like schizophrenia.

Management

Pharmacotherapy

It is a commonly observed trend in general practice, to prescribe benzodiazepines such as alprazolam to depressives in order to improve their sleep since many patients believe that they will get all right once their sleep improves. It should be remembered that these drugs have no action on the core symptoms of depression, they bring about only an initial and temporary improvement in sleep and are, therefore, of no use alone in the treatment of depression. Antidepressant drugs are the treatment of choice since they work on the specific neurotransmitter abnormalities that cause depression. The dose requirement varies from person to person. The drugs start working after about 3-4 weeks of starting the treatment.

A list of commonly prescribed antidepressant drugs is given below

Generic name (mg/day)	Starting dose (mg/day)	Daily dose
Tricyclics:		
Imipramine	25-50	100-300
Amitriptyline	25-50	100-300
Doxepin	25-50	100-300
Dothiepin	25-75	150-300
Clomipramine	25	100-250
Nortriptyline	25	50-200
SSRIs:		
Fluoxetine	20	20-60
Citalopram	20	20-60
Sertraline	50	50-200
Paroxetine	20	20-60
Fluvoxamine	50	50-300
Other newer drugs:		
Amoxapine	50	100-400
Venlafaxine	37.5	75-225
Mirtazapine	15	15-45

Drugs have to be continued even after all the symptoms of depression have abated. A maintenance course has to be given for at least 6 to 9 months for complete remission.

The commonest side effects of the tricyclic group of drugs are dryness of mouth, constipation, blurring of vision, postural hypotension, and urinary difficulty and in very high doses, cardiotoxicity. The SSRI group of drugs usually cause nausea, abdominal discomfort, tremors, restlessness and insomnia. Amoxapine can cause extrapyramidal symptoms. Venlafaxine can cause dizziness, a rise in blood pressure, constipation and abdominal discomfort. Mirtazapine can cause excessive sedation. Generally, all the side effects occur in the initial stages and can be tackled with the help of other appropriate measures e.g. giving a laxative for constipation; and with proper psycho education of the patient and the family.

Referral to a psychiatrist

It is not advisable to wait for antidepressant drugs to act when the patient is suicidal, especially if the patient has psychomotor agitation. In such cases, it is preferable to hospitalize the patient and start electroconvulsive therapy. Generally, about 8 to 12 sessions of ECT given on alternate days bring about a rapid change in the clinical picture. Drugs also have to be started simultaneously and continued further.

Psychosocial intervention

Psychotherapy is a very useful adjuvant to the above-mentioned treatment methods. It helps the depressive patient to overcome psychological problems such as low self-esteem, inferiority and problems pertaining to different life situations such as the family, workplace etc.

Conclusion

Depressive disorders are among the commonest mental disorders seen in clinical practice. It is important for a clinician, therefore, not to miss the diagnosis since the quality of life of these persons is severely affected and it can lead to life-threatening complications such as suicide. Depression is likely to be missed in patients who do not verbalize their symptoms but somatize them i.e. have physical symptoms. Such patients often undergo unnecessary physical investigations and are finally told that there is nothing wrong with them. This may be devastating statement for the patient to accept who is suffering a lot. Such patients need an explanation that their physical symptoms have no physical cause and that they have to be treated differently. A warm, supportive approach is essential in order to manage these patients effectively. Early diagnosis and adequate treatment are the key words in the management of depression.

Mixed Anxiety and Depressive Disorder

N. G. Desai
Deepak Kumar
D. K. Gupta

Case of worried/ anxious depressed

Mrs. Lata, a 32 year old housewife presented at the primary health center, with persistent psychiatric symptoms for the past seven months. She had difficulty falling asleep and would keep tossing and turning in her bed. There was no temporal correlation with any stressful life event. She would seem to be worrying, irritable, and complaining of sad mood. She reported headache and vague body pains and palpitations. She also felt worthless and was low in her self-confidence. On further enquiry, there was no history to suggest any general medical condition or history of drug abuse, obsessions, or phobias. There was no associated suicidal behaviour, psychomotor retardation, or cognitive decline. Patient was distressed and concerned over her symptoms and was not able to carry out her day-day work as efficiently as before and hence sought treatment with a physician at the primary care setting, considering that she might be having some medical problem related to head or heart. However, physical examination and suitable investigations did not reveal any abnormality.

If we analyze the clinical history of Lata, we find that she had depressive symptoms like sad mood, poor sleep, depressive thoughts of worthlessness and low self-confidence; as well as anxiety symptoms like worry, palpitations and vague muscular aches. Due to these symptoms she also has significant distress and functional impairment. Hence she could be diagnosed as a case of Mixed Anxiety and Depressive Disorder (MADD). Thus, MADD should be diagnosed when a patient has concomitant symptom of anxiety and depression.

Mixed anxiety and depressive disorder

Anxiety and depression often coexist in clinical settings. Substantial overlap of both symptoms creates diagnostic dilemmas. Recognition of anxiety and depressive disorders in primary care settings is now considered to be extremely important in health care delivery. It has been observed in primary care settings as well as in epidemiological studies that there is a group of patients, having subsyndromal anxiety symptoms and subsyndromal depressive symptoms that are not sufficient to warrant diagnosis of either an independent anxiety disorder like GAD or depressive disorder like major depression. However, these patients have distress and impaired functioning which necessitates categorizing them having a psychiatric illness and treating them. In the recent times the recognition of mixed anxiety and depressive disorder as a separate diagnostic category has led its inclusion in ICD-10, and also being proposed for DSM-IV classification.

Epidemiology

The epidemiological studies carried out so far reveal varying figures both in primary care as well as general population settings. The wide variation in estimates is because of the fact that different studies have not defined cases in a similar way. The prevalence of MADD in primary care settings is reported between 4.1% (Barrett et al., 1988) to 12.8% (Stein et al., 1995). However the prevalence figures in general population is reported between 0.29-0.8% (Wittchen et al., 1993). It is important to understand that majority of these patients were unrecognized by general practitioners and those who were diagnosed were not being adequately treated. These are western figures and corresponding Indian data are not available, though the clinical experience indicates the presence of similar trends in India.

Mixed anxiety and depressive disorder is a common condition encountered in routine clinical practice with a greater preponderence in females compared to the male population.

Clinical features

Mixed anxiety and depressive disorder is a new diagnostic category recently introduced in the ICD-10 classificatory system for patients seen mainly in primary care settings. This is a condition characterized by both anxiety and depressive symptoms of limited but equal intensity but is not sufficient to fulfill the criteria for specific anxiety or depressive disorder, and which is independent of stressful life events. It is often encountered in primary care and community settings, where the distinction between anxiety and depression is difficult and sometimes impossible to make. Hence, there is clearly a need of awareness of this condition for physicians and medical practitioners.

A patient presenting with both anxiety and depressive symptoms can be diagnosed as a case of mixed anxiety depressive disorder if he has persistent or recurrent dysphoric mood in the previous months (at least one month) accompanied by following symptoms (minimum four)

1. Difficulty concentrating or mind going blank
2. Sleep disturbances (difficulty falling or staying asleep, or restlessness, unsatisfied sleep)
3. Fatigue or low energy
4. Irritability
5. Worry
6. Being easily moved to tears
7. Hypervigilance
8. Anticipating the worst
9. Hopelessness (pervasive pessimism about the future)
10. Low self-esteem or feelings of worthlessness

The above symptoms must cause clinically significant distress or impairment in social, occupational or other important areas of functioning. The symptoms should not be due to direct physiological effects of a substance (e.g. a drug of abuse, a medication) or a general medical condition. The symptoms should not be severe enough to meet the diagnosis of any of anxiety or mood disorders. He should not have met the criteria for major depressive disorder, dysthymic disorder, panic disorder, or generalized anxiety disorder or any other psychiatric disorder in the past. Thus, the clinical profile of MADD involves both the psychic as well as somatic symptoms pertaining to both anxiety and depression.

Differential Diagnosis

The key consideration in clinical evaluation of such patients is that symptoms of a more severe disorder are not missed. They need to be differentiated from "pure" anxiety /depressive disorders, somatoform disorders, chronic physical illnesses with minor psychological symptoms.

Anxiety and depression are commonly seen together in clinical practice. Mixed Anxiety and depressive disorders (MADD) is a recent diagnostic entity to be recognized in general practice. There has been a lot of debate regarding its etiology, diagnostic validity, course and prognosis .The points

relevant to primary health care settings are as follows:

- Major Depression/ Dysthymia/ GAD/Panic disorder should not be diagnosed as MADD.
- Minor symptoms of anxiety and depression in patients with physical illnesses to be differentiated from MADD.
- The somatoform disorder group of patients also should be differentiated and not misdiagnosed as MADD.
- MADD has overlap with adjustment disorder. Chronicity, recurrent nature and absence of relation with stressful life event differentiates the former from the latter.

Course and Prognosis

MADD has been seen to manifest almost at any age during the lifetime. MADD is associated with mild to moderate but significant dysfunction in occupational, social and family spheres (Roy-Byrne et al., 1996). Although there is paucity of literature on the course and outcome of this clinical condition, clinical experience testifies that majority of the patients benefit from psychiatric intervention.

Management

Pharmacotherapy

In view of paucity of data on treatment for MADD, the clinicians often use approaches that are effective in other mood or anxiety disorders. There is no consensus regarding the treatment of MADD, with uncertainty shrouding in this regard - whether this is an anxiety accompanied by depression that should be treated with anxiolytics, such as benzodiazepines or is this a depression accompanied by anxiety that should be treated with an antidepressant. Tricyclic antidepressants (TCAs) like imipramine and amitryptyline have been found to be effective in anxiety symptoms besides depression but at the cost of distressing side effects. Dothiepin, another TCA has been found to be effective and widely prescribed in MADD in doses of 50-100 mg/day.

Studies so far have reported that benzodiazepines, although useful, do not provide effective treatment for depressive symptoms. Increasingly, SSRIs and newer antidepressants like venlafaxine are being used with good results in both sets of symptoms (Beck et al., 2000). SSRI's especially fluoxetine, sertraline and citalopram have been found to be effective in this disorder.

Thus pharmacological treatment in the form of antidepressants (Dothiepin, SSRIs, Venlafaxine) instead of BZDs are being advocated. The usual dose required for MADD is less than that required for major depression. BZDs are widely prescribed by general practitioners for patients with MADD based on the fact that anxiety symptoms are more likely to be reported spontaneously and are readily observed in clinics. However, there is a risk of producing iatrogenic dependence on BZDs.

Psychosocial interventions

Various non-pharmacological treatment modalities like relaxation therapy and psychotherapeutic interventions like brief counseling and cognitive behaviour therapy in suitable cases need to be supplemented to the drug regime for better results.

Conclusions

Mixed anxiety depressive disorder has been identified as a new diagnostic entity. It is a specific disorder and is included as a separate category in ICD-10 (and has also been proposed for DSM-IV) classificatory system in view of significant dysfunction associated and its high prevalence in primary care settings. It has been found to be a treatable condition with pharmacological as well as non-pharmacological interventions. The adequate knowledge about MADD among general practitioners is of vital importance.

Adjustment Disorders

B.M. Suresh
C.R. Chandrashekar

Case of fracture and its consequences

Mr.Sujeet, was a 36 year old married gentleman, working as a mechanic in a private factory. He was very energetic with high aspirations in life and a pleasant disposition. He lived in a nuclear family consisting of his wife and two daughters. One day while returning from his factory, Sujeet met with an accident, during which he sustained a crush injury to his right leg. He was taken to a private hospital where the orthopedician and the vascular surgeon tried to do restorative surgery. Unfortunately gangrene set-in after the emergency operation. Hence the specialist decided to go in for a below knee amputation. The patient refused to consent for the surgery but later on, left with no options, he agreed for it. Operation went on without any complications. Postoperative-period was uneventful and he was discharged within a week.

A fortnight after the operation on follow-up Sujeet was brought with complaints of feeling uneasy, having decreased sleep, multiple body aches, being withdrawn, irritability on trivial issues, anger out bursts, reporting fear at times, decreased interest in pleasurable activities and occasional crying spells. On one occasion he expressed death wishes to his wife. The orthopedician did not find any post-operative complications. During the consultation Mr. Sujeet started crying and asked the orthopedician for some injection, which would put an end to his life without any pain. On hearing his suicidal ideation the orthopedician referred the patient immediately for psychiatric consultation. Psychiatric evaluation revealed that his symptoms had increased since two days when he came to know from his colleague that he might lose his job. History of marital conflicts was also revealed. During the therapy he said, 'I can't return to my old job, I am worthless. It's not fair that I am subjected to such punishment. I am a burden on my family. No one really cares for me'.

Taking into consideration the clear-cut stressors and presentation of both anxiety and depressive symptoms of short duration, a diagnosis of 'Adjustment Disorder with Mixed Anxiety and Depressed mood' was made.

Supportive psychotherapy and Tab. Buspirone 10mg twice a day was started to take care of his anxiety symptoms. His disabilities, the possibility of losing his job, alternative ways of coping with this situation, and his marital problems were discussed. He was persuaded to be hopeful. Within 2-3 days Sujeet started feeling better and denied suicidal ideas or death wishes. He was sent for physiotherapy where he started interacting with the other patients and realised that he was not the only unfortunate but there were many others. His wife was advised to give him support and assure him that she would stand by him in facing the future. Then he received intensive physical therapy to strengthen the stump and learn how to use prosthesis. Within a few months he attained premorbid level of functioning and got back to a job.

Adjustment disorders

The development of transient psychiatric symptoms in the context of stress is virtually a universal experience. An adjustment disorder is defined as development of emotional or behavioral symptoms in response to an identifiable stressor(s) occurring within a month of the onset of the stressor(s) and the duration of symptoms usually does not exceed 6 months, except in the case of a prolonged depressive reaction. These symptoms or behaviors are clinically significant as evidenced by either of the following:

1) Marked distress that is in excess of what would be expected from exposure to the stressor.
2) Significant impairment in social, occupational or educational functioning.

Stressful event might be

- The presence or possibility of serious physical illness. (Disabling or Life threatening)
- Job loss, business loss
- Parental divorce, separation, bereavement
- Migration or refugee status
- Breaking up with spouse or partner
- Child being put to a new school
- Failure to reach the goal

Epidemiology

Adjustment disorder is very common. Prevalence of an adjustment disorder in general population is 1-2%. Depending upon the population studied prevalence of adjustment disorders varies. Studies done on clinical population report a wide range of prevalence of adjustment disorder from 15-95%.

Adjustment disorders affect both genders equally. Adjustment disorders can occur at any age. People are particularly vulnerable during normal transitional periods such as adolescence, mid-life and late life.

Pregnancy and the puerperium are vulnerable periods in every woman's life. 5-8% had diagnosis of adjustment disorder during that period (Knop, 2001).

Adjustment disorders have been reported frequently in individuals with a variety of medical disorders like vitiligo, psoriasis, cancer, diabetes mellitus etc., (Mattoo et al., 2001; Akechi et al., 2001; Prieto et al., 2002; Kovacs et al., 1995).

Etiology

Adjustment disorders are a reactions to stress, but there is no way to predict the individuals who are likely to get an adjustment disorder given the same stressor. Patients with adjustment disorder have increased vulnerability to stress. This vulnerability depends upon patient's life values, adaptation level, personality, past stress experiences and coping skills. Diminished psychological defenses and inability to cope with stressors on a cognitive level lead to suicidal behavior in patients with adjustment disorder. (Polyakova et al., 1998)

Stress, frequently described, as 'the Black Plague' or 'the Modern Epidemic' has become an important feature and major problem of everyday life, threatening individual, organisation and societal health. Hans Selye, who is often referred to as 'father of stress research', has defined stress as the 'non-specific response of the body to any demand', the General Adaptation Syndrome (GAS).

Lazarus (1966) postulated that an individual's perception of stress was significantly more important than the event per se in determining the impact of the stressor. Stressors also vary in duration, intensity and effect. Impact of stress on an individual is modified by a number of intrinsic factors (e.g.,

genetic vulnerability, premorbid personality) and extrinsic factors (e.g., social support). Currently there is no evidence available to suggest a specific biological factor that causes adjustment disorders. We know that stress leads to various physical and mental illnesses as it has also been proved beyond doubt that stress leads to poor outcome of any physical illness and mental illness. (Illescas-Rico et al., 2002, Goodyer et al., 1987).

Clinical features

The adjustment disorders in the ICD-10 have been placed in a cluster of Stress-related disorders. As per the diagnostic criteria:

- The onset of symptoms must occur within 1 month of exposure to an identifiable psychosocial stressor, not of an unusual or catastrophic type
- The individual manifests symptoms or behavior disturbances, which should not meet the criteria for any other mental disorder

People with adjustment disorder may have a wide variety of symptoms. How those symptoms cluster depend on the particular subtype of adjustment disorder and on the individual's personality and psychological defenses. Adjustment disorders has been classified depending upon the type of presentation.

There are seven subtypes of adjustment disorder that are based on the type of the major symptoms experienced. The following are the most common symptoms of each of the subtypes of adjustment disorder. However, each adolescent may experience symptoms differently. Symptoms normally include some (but not all) of the following

1) **Brief depressive reaction** is a transient mild depressive state of duration not exceeding 1 month. Lack of interest in usual activities, withdrawal, inhibition, including loss of appetite or interest in sex, hopelessness, sadness, crying, feelings of loss, suicidal ideas/attempt, guilt and self-doubt not severe enough to warrant the diagnosis of other psychiatric disease like depression, GAD etc.

2) **Prolonged depressive reaction** is a mild depressive state occurring in response to a prolonged exposure to a stressful situation but of duration not exceeding 2 years.

3) **Mixed anxiety and depressive reaction** is characterized by anxiety symptoms like breathing fast, numbness, heart pounding, excitement, fear, trembling, nausea, being easily startled by loud noises or sudden movements, worrying, feeling unreal, being isolated or detached from other people, problems with thinking, concentration or remembering things, preoccupation with the stressor, sleep problems including difficulty in getting sleep, waking in the middle of the night, dreams or nightmares. Both anxiety and depressive symptoms are prominent, but at levels no greater than those specified for mixed anxiety and depressive disorder or other mixed anxiety disorders.

4) **With predominant disturbance of other emotions:** The symptoms are usually of several types of emotions, such as anxiety, depression, worrying tensions, loss of feelings, irritability and anger. This category should also be used for reactions in children in whom regressive behavior such as bedwetting or thumb sucking may be present.

5) **With predominant disturbance of conduct:** The main disturbance is one involving conduct, e.g., an adolescent grief reaction resulting in aggression, irritability, dissocial behavior like violation of the rights of others or violation of normal societal behaviors and rules (truancy, destruction of property, reckless driving, fighting) etc.

6) **With mixed disturbance of emotions and conduct:** Combinations of symptoms from all of the above subtypes are present (depressed mood, anxiety, and conduct).

7) **With other specified predominant symptoms:** Like unexplained physical symptoms such as tense muscles, trembling or shaking, diarrhoea or headaches, stomachaches, constipation, nausea, headaches, sweating, tiredness.

Diagnostic Guidelines

Diagnosis depends on a careful evaluation of the relationship between

- Form, content, and severity of symptoms
- Previous history and personality and
- Stressful event, situation, or life crisis. The presence of this third factor should be clearly established and there should be a strong, though perhaps presumptive, evidence that the disorder would not have arisen without it. It is important ti note that adjustment disorder, in children includes grief reaction and hospitalization.

Grief and Bereavement

Loss is a universal experience. During life everybody has to cope with a series of losses, which may range in intensity from the minor loss of a pet to the major loss of a spouse or child.

Bereavement is the loss of a loved person through death. Grief stands for the feelings and associated behavior (e.g., crying) precipitated by this death. Mourning indicates the social expression of grief, including funerals, visitations and rituals.

Stages of normal grief

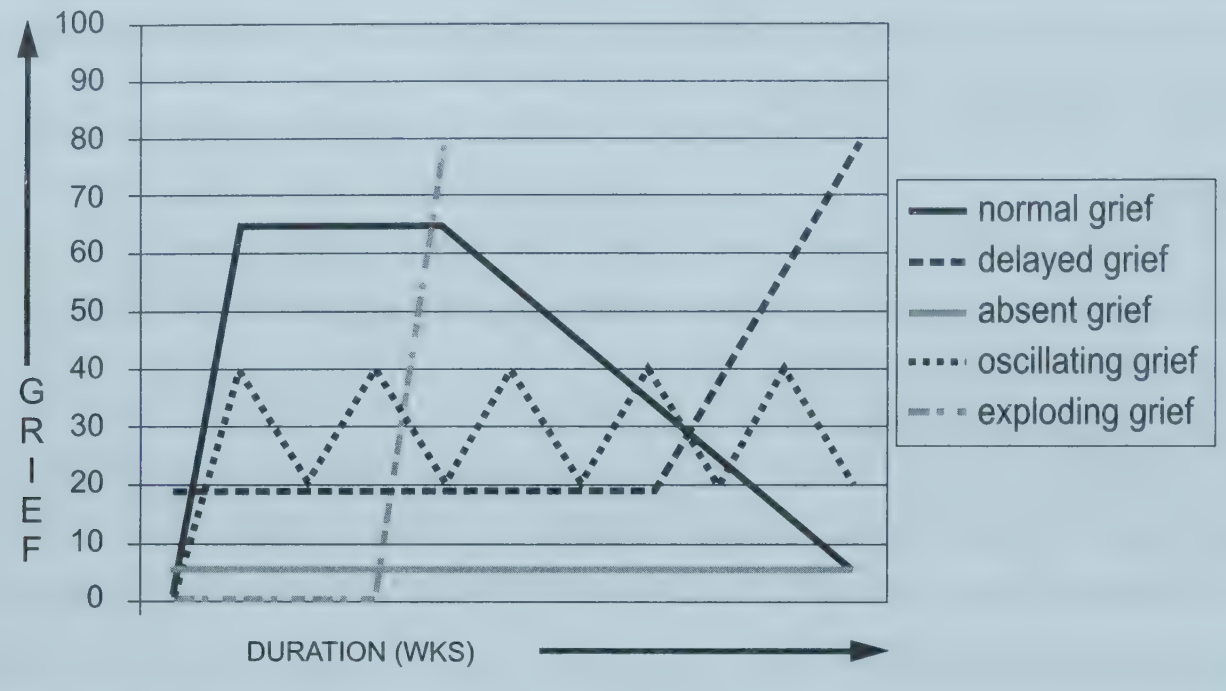
1. A first phase of shock, disbelief, numbness and denial followed by searching behaviors lasting for days to weeks.
2. A second phase of acute anguish characterized by intense somatic distress, preoccupation with the deceased, anger, restlessness, bargaining, feeling of sadness, guilt, and impairment in functioning lasting for weeks.
3. A third phase of restitution and reorganization (acceptance) lasting for a few months.

Normal grief usually follows the above phases with a possibility of some variation. It is important to identify abnormal grief, as if undetected, it could lead to various problems like non-acceptance of the death of the individual, avoidance of the mention of the deceased, avoidance of the funeral and other associated rituals, holding on to the belongings of the dead, preoccupation with thoughts of the deceased, a strong and continued feeling of the dead person's presence, illusions or hallucinations of the deceased for a long time.

Abnormal grief can be grossly classified into the following

- Delayed grief
- Absent grief
- Oscillating grief
- Exploding grief

Graph depicting patterns of normal and abnormal grief reactions



Course and Prognosis

Adjustment disorder occurs in response to an identifiable stressor. The person's response to the stressor is either very strong and causes a great deal of distress (feeling suicidal after losing a limb in an accident) or results in impairment in functioning in either the person's occupational/education, social or interpersonal life (i.e. missing work for a week after breaking up with a boy/ girl friend). Still others may not experience behavioral disturbances, but will begin to suffer from physical illness. If someone is already suffering from a medical illness, that condition may worsen during the time of the adjustment disorder. Most people recover completely from adjustment disorders. People with no history of prior psychiatric symptoms, and who have stable environments and strong social support are likely to return quickly to their premorbid functioning level. The majority of these people will have a very positive prognosis, and are likely to display no further psychological symptoms in the future. But people with progressive or cyclic disorders (such as multiple sclerosis) may experience an adjustment disorder with each exacerbation period.

Kovacs et al 1994, reported a 76% recovery rate within a year, which was maintained even after a 10 year, follow-up and also it did not predict later dysfunction. Adjustment disorder should be predicted as having a relatively good outcome and most patients should be well at follow-up.

Management

The primary goals of treatment are to relieve symptoms and assist with achieving a level of adaptation that is comparable to the affected person's level of functioning before the stressful event.

Pharmacotherapy

Most acute stress reactions will resolve without the use of medication. Doctor's empathetic reassurances are as effective as benzodiazepines. However, if severe anxiety symptoms occur, consider using anxiolytic drugs for up to ten days. If the patient has severe insomnia, use hypnotic drugs for up to ten days. Physicians should be especially careful of over-prescribing possibly addicting medications for anxiety. Doses should be as low as possible (Michael et al., 1993).

Short acting benzodiazepines like Lorezpam in the dose of 1-4 mg/day can be prescribed. Long acting drugs like Diazepam, Nitrazepam and Chlordiazepoxide have to be used with caution.

Buspirone, acts as an anxiolytic without any side effects or addictive property like other sedatives. The usual initial adult dosage is 10-15 mg/day in three divided doses. Patients may benefit from dosage titration up to 30-60 mg/day. While medications have very limited value in the treatment of adjustment disorders, medication may be considered on a short-term basis if a specific symptom is severe and known to be responsive to medication.

Strong suicidal ideas or attempt

Suicidal ideas or attempts are one of the common presentations in adjustment disorder (Kryzhanovskaya & Canterbury, 2001; Polyakova et al., 1998). It is a psychiatric emergency, calling for immediate psychiatric referral.

Psychosocial intervention

Individual Psychotherapy

Psychotherapy (counseling) is the treatment of choice for adjustment disorders, since the symptoms are an understandable reaction to a specific stress. Adjustment disorder responds better to psychotherapy than medication (Andreasen & Wasek, 1980). The type of therapy depends on the health professional, but it usually is short-term treatment that focuses on resolving the immediate problem.

Therapy should occur within a supportive, non-judgmental environment that encourages the client's growth through exploration of new behaviors and ideas. This therapy often takes the form of solution-focused therapy, to help the individual deal more effectively with the specific life problem. Often the therapist acts as a partner in therapy, helping and guiding the client towards finding these new coping mechanisms, or finding a better understanding of issues and problems.

Adjustment disorder, by definition, is a shortterm difficulty that rarely goes beyond 6 months. Lingering feelings may occur beyond that time, but those are natural and not likely to be severe enough to require additional attention or treatment. It often helps treatment progress (and is required in many agencies) to put together a firm but realistic treatment plan, so that the patient can also see the short term nature of the therapy. Clinicians should be careful not to lapse into acting as an advice-giver to individuals who suffer from an adjustment disorder.

The exact content and type of therapy used will vary widely. Treatment will often emphasize the importance of social support within the client's life, alternative activities to explore or to find meaning in, increasing a person's range and effectiveness of coping skills, learning better ways of dealing with stress, etc.

Group Psychotherapy

Group Psychotherapy is useful if similar group of patients are involved in the therapy. For example group therapy done once in a month in patients under going treatment for early stage breast cancer was proved to be effective (Hosaka et al., 2001). Before approaching the concrete work of planning and organizing a psychotherapy group, the goals of the group must be clearly understood and developed by the leader. These in turn, depends on the setting, the population, the time available for treatment, and the training and capacity of the leader or leaders. Therapy groups can be organized with a variety of goals in mind like character change, supporting homogeneous patient populations, targeting certain symptoms, reestablishing premorbid levels of functioning etc.

A major advantage in group psychotherapy is that patients feel a sense of belonging. Most people enter group therapy feeling that their problems are uniquely shameful and that their pathology sets them apart from competent human beings. Group members come to recognize that their problems are more similar than different from those of others in the group, as they increasingly feel accepted and cared for.

Working with the family of the patient

Give essential information to the patient and family, regarding illness. Identify relatives, friends and community resources, who are able to offer support. Encourage the patient to acknowledge the personal significance of the stressful event. Shortterm rest and relief from stress may help the patient. Consider shortterm sickness certification. Encourage a return to usual activities within a few days to weeks.

Family therapy is often focused on making needed changes within the family system such as improving communication skills and family interactions, as well as increasing family support among the family members. Family therapy may be appropriate for certain individuals, especially if the person presenting with the disorder is an adolescent. This type of therapy also is appropriate when the family is 'scapegoating' a particular family member, or there is a clear 'identified patient', when the actual problem is family-systems related. Education related to the disorder is sometimes needed, and the family can be reassured as to the nature and seriousness of the disorder, as well as its prognosis. Couples therapy is appropriate when the disorder is additionally negatively affecting the marital relationship.

Self-help groups

Self-help methods for the treatment of this disorder are often overlooked by the medical profession because very few professionals are involved in them. Often people with this disorder will get help from attending a group related to their specific problem. This could be anything, ranging from someone who just got divorced, to someone who was just diagnosed with cancer, to dealing with job loss, etc.

Many such support groups exist in communities across the nation, so finding an appropriate one may not be difficult. This allows for the sharing of information and experiences, which can be vital in the road to recovery. Social support is also a vital component of a self-help group and increased social support usually leads to better and quicker recovery.

As an adjunct to regular psychotherapy, people can also be encouraged to use a support group to try out new coping skills and express their feelings to others who have gone through similar experiences. This is usually very rewarding and helpful.

Management of Grief

In general, the rituals associated with death serves the purpose of mourning. These rituals afford an opportunity for catharsis and relieving, which help the bereaved in getting over the loss. The judicious use of drugs does not hinder grieving, but helps in overcoming the abnormal/ excessive emotions during abnormal grief. The drugs should be used cautiously depending upon the type of presentation.

The essence of treatment is however psychosocial intervention in the form of grief therapy. This essentially involves getting the individual to talk about the deceased, recollecting the experiences preceding and succeeding the death, ventilating his/her feelings both right and wrong towards the dead person and finally letting go of the deceased. During the sessions the therapist plays a supportive role and helps in rationalizing the patients feelings towards the dead one. It would help to bear in mind that during the process of grief therapy the depressive symptoms of the patient may escalate or worsen for a brief period indicating that the person is grieving. It is at this stage that the patient requires the support of the therapist the most and hence the therapist should take care not to abort or digress from the therapy. Time-limited, short-term group therapy is one of the successful approaches in complicated grief (Piper et al 2001).

Conclusion

Since incorrect diagnosis may result in inadequate treatment, inappropriately conceptualizing a patient's problem as constituting an adjustment disorder may result in delays or inaccuracies in treatment planning. Misdiagnosis of other, more specific disorders when an adjustment disorder should appropriately be diagnosed is also a problem. When treating a patient, diagnosed to be suffering from adjustment disorders, one needs to be aware, that pharmacological intervention in this population should be used to augment psychosocial strategies rather than serving as the primary modality.

Obsessive Compulsive Disorder

Sumant Khanna

Case of obsessive compulsive disorder

A 32 year old married female with 2 children presents with a 7 year history of repeated washing. The illness started soon after the delivery of her second child. There is no other contributory family, personal or past history. When the illness started she would get doubts that her child would become dirty because of her; would get an infection and may die. Because of these thoughts she would spend an excessive amount of time washing her hands and reassure herself that she was not dirty. Both the frequency and the amount of time she spent each time washing her hands was excessive. Gradually these thoughts started involving others in the family and she repeatedly started washing the food she was cooking, to ensure that it was clean. This started resulting in delayed completion of household chores and disruption of the routine of other people at home. As her children grew older, she started insisting that they have a bath immediately after returning from playing outside, and she would beat them if they refused. Initially she used to use a single soap over a period of two to three days, but later shifted to 1-2 liquid soap bottles per day. She recognized these thoughts were her own thoughts, but they used to occur repetitively and intrude on her conscious awareness without her will. She was unable to prevent these thoughts, and if she was not able to engage in the washing would become very anxious. Because of these thoughts she started feeling that something was drastically wrong with her and she should not live. She would feel hopeless and worthless because of these thoughts, over the last 3 years. Over the last 3 years she has also restricted going out of the house because she felt that she could thereby avoid getting dirty and infected. She would repeatedly wash the floor with phenol to ensure that it was clean and not dirty. These washing rituals would take up the better part of the day. She believes that there is nothing wrong with her except for the fact that she gets these thoughts, which she should be able to control on her own, and is not keen on medical intervention, which has contributed to the long gap between onset of symptoms and consultation. She has been reluctantly, brought by her relatives on the advice of their general practitioner.

The symptoms she is suffering from are repetitive, intrusive thoughts about contamination and getting infections, which she recognizes as being senseless and tries to resist but is unable to do so. To decrease the anxiety she engages in, and gets others to engage in, various ritualistic activities. These symptoms are classical obsessions and compulsions, and she is suffering from Obsessive Compulsive Disorder (OCD). She also has secondary depression. Although she has social avoidance, this is part of OCD and does not meet criteria for Social Phobia.

She would benefit from a combination of Selective Serotonin Reuptake Inhibitors and Behaviour Therapy. The drugs would take at least 8-12 weeks to act. Behaviour therapy would focus on exposure to the obsessive themes involving contamination and getting an infection, and response prevention as far as her compulsions regarding washing are concerned. She would have to be explained the rationale for her treatment plan, and convinced to engage in behaviour therapy. With this treatment package she would achieve 70-90 % reduction in symptoms, and would have greater ease in dispelling these thoughts regarding contamination and illness, even though these thoughts would still occasionally occur.

Obsessive Compulsive Disorder

Obsessive compulsive disorder is an intriguing and often disabling syndrome characterized by two distinct phenomenon: obsessions and compulsions. Although the syndrome has been well described since the fifteenth century, it was thought to be relatively rare. However the Epidemiological Catchment Area study in 5 sites in USA established it as being a fairly common syndrome with a prevalence of over 2%. This fuelled a lot of interest and study in the treatment and pathogenesis of this disorder over the last 2 decades (Khanna and Venkatsubramaniam, 2003; Khanna, 1999).

Epidemiology

The results of a large psychiatric epidemiological study, the national ECA Survey, conducted in the United States in 1984 found that OCD was the fourth most common psychiatric disorder. It had a 6 month point prevalence of 1.6% and a lifetime prevalence of 2.5%. Similar findings have emerged from studies conducted in Puerto Rico, Canada, Germany, New Zealand and Korea; however lower rates have been reported from Taiwan and India.

Women appear to develop OCD slightly more commonly than men; however there is a strong predominance of males in a pediatric population. Women more often seem to have washing rituals as compared to men, and a later age of onset. The childhood onset group seems to have a higher genetic mediation as compared to adults, a finding replicated in the Indian setting.

Etiology

Both biological and psychosocial factors seem to play a role in the genesis of OCD. Genetic factors appear to predispose to development of OCD in some individuals, but genetic factors do not yet account for all cases. Several neurotransmitter systems have been implicated in the etiology of OCD but a deficient serotonergic status seems to be the most consistent observation. These dysfunctions do not seem to be generalized but limited to certain parts of the brain, namely the pre-frontal cortex, the cingulate gyrus and the caudate nuclei. There appears to be a disturbed circuit connecting these parts of the brain, and changes in these areas have been seen with effective therapies (Rauch et al., 2002).

Psychosocial factors play a smaller role in the genesis or maintenance of the disorder. OCD was one of the first disorders thought to have a psychosexual origin according to Freudian thought, and learning principles underlie the practice of effective behavioural therapies. Most life-events other than pregnancy do not seem to have an impact on the onset of OCD, although family factors are involved in the maintenance of the disorder. A sub-group of patients seem to develop the disorder as an auto-immune response to streptococcal infection.

Clinical features

Obsessions (intrusive, inappropriate and disturbing ideas, thoughts or images) and compulsions (repetitive behaviours to reduce anxiety) are the core symptoms of OCD. The Yale-Brown Obsessive Compulsive Check-List has been derived from clinical experience. The obsessive categories are aggressive, sexual, religious, somatic, symmetry, contamination, hoarding and others; the

compulsive categories are checking, ordering and re-arranging, counting, repeating rituals, cleaning, hoarding and collecting, and others. The most common obsessions is a fear of contamination, followed by pathological doubt, somatic obsessions, and need for symmetry. The most common compulsions are checking, followed by washing, counting, need to ask or confess, and symmetry and precision. Children with OCD frequently have magical thinking, which extends beyond their normal developmental age frame (Eisen & Rasmussen, 2002; Khanna, 1990).

Most patients with OCD have multiple obsessions and compulsions over time, with different symptoms being predominant at different times. About a third of patients do not have compulsions; there is a need to look out for reassurance rituals or unrecognized mental compulsions (such as repetitive, ritualized praying). Pure compulsions are relatively rare, except in children.

Traditionally the awareness of senselessness or unreasonableness of obsessions (often referred to as insight) and the accompanying struggle to overcome the obsessions (referred to as resistance) have been regarded as being essential to the diagnosis of OCD. However numerous descriptions of patients who are completely convinced of the reasonableness of their beliefs and the need to perform compulsions have appeared in the literature. Systematic studies have shown that insight is not an all or none phenomenon, and varying degrees of insight can be present, and this is not a predictor of treatment response. DSM-IV has established a new OCD specifier: 'with poor insight', for situations when, for most of the time, the individual does not recognize that the obsessions or compulsions are excessive or unreasonable.

Differential diagnosis

Although obsessions and compulsions are classical and characteristic of OCD, at times there are issues regarding the phenomenon itself, which need to be clarified. Also obsessions and compulsions can often co-exist with other psychiatric conditions, notably mood disorders, anxiety disorders and psychosis.

The issue of OCD with pure insight, often raises the issue whether the phenomenon is an obsession or a delusion; the intrusiveness and repetitiveness are the keys to answer this dilemma. One of the older beliefs that the severity of the condition and the disability that it causes may also help differentiate, no longer holds true. There is a phenomenological overlap with phobias, which are recognized as irrational fears with avoidance behaviours, and similar symptomatology in OCD. Phobias tend to be focal, specific and stable over time; obsessive compulsive phenomena are usually multiple and tend to change over time. Because of shared therapeutic strategies, the differentiation is probably academic, but the co-occurrence of these two syndromes is also frequent. Subjects with anankastic personality disorder also have patterns of behaviour which are long standing, and have low insight and resistance. The relative lack of disability and the absence of overt-obsessions and compulsions are key issues in differentiating these diagnosis.

Comorbidity

Mood Disorders

Depression is the most common comorbid syndrome. In over 90% of cases it develops after OCD and is recognized as being secondary depression. A small minority of patients have onset of depression

before or with their obsessive compulsive symptoms. It is difficult at times to differentiate between primary depression and depression secondary to the demoralization and hopelessness accompanying OCD symptoms. A few patients view their depression as being independent of their OCD. The co-occurrence of Bipolar disorders with OCD, which was initially thought to be rare, is now getting more importance, and poses a major therapeutic challenge.

Anxiety Disorders

Other anxiety disorders frequently co-exist with OCD. A comorbidity of 10% of OCD with panic disorder, social phobia and/or generalized anxiety disorder has been reported. Conversely relatively high lifetime rates of social phobia, panic disorders and specific phobias have been reported in OCD probands. The high prevalence of anxiety states in these patients may be caused by common developmental and temperamental traits whose phenotypic expression is secondary to shared genotypic and psychosocial factors.

Tic disorders

About a third of patients with Tourette's disorder have OCD, while about 20% of OCD probands have a lifetime history of multiple tics. Comorbid OCD and Tourette's syndrome occur at an earlier age, and have family pedigrees, which are loaded for both disorders. Some symptoms like symmetry and ordering are also more frequently seen in this population.

Eating disorders

A history of anorexia nervosa has been reported in about 17% of patients with OCD. Phenomenological similarities exist between the anorexic's preoccupation with body image and obsessive compulsive symptomatology.

Schizophrenia

Studies have shown comorbidity of OCD and schizophrenia in 8-40% of the sample studied, based on the methodologies used. The Chestnut Lodge follow-up study showed that the co-morbid disorders had a more chronic course and a greater degree of social and occupational impairment. Neuropsychological deficits in visuo-spatial skills, delayed non-verbal memory and cognitive shifting abilities were also worse in the combined group. Comorbid OCD and schizotypal disorder has a poorer response to treatment, both pharmacological and behavioural.

Personality disorders

The most common personality disorders encountered in OCD are anxious-avoidant, dependent, passive-aggressive and obsessive compulsive. Schizotypal, paranoid and borderline personality disorders are found less commonly, but seem associated with a poorer outcome. Although obsessions and obsessionality, were initially thought to be interminably linked, this has not been confirmed by systematic studies. A discontinuity between the two syndromes is clearly reflected in the diagnostic criteria in use today.

Course and Prognosis

The mean age of onset is around 20 years, with males having a younger age of onset. Fewer than 15% had an onset of illness after 35 years. Most patients with OCD have a chronic course with some fluctuations in the severity of symptoms over time. Although initially thought to have a poor outcome, the advent of behavioural and pharmacological therapies has led to substantial improvement in a number of patients; while complete remissions are unusual. An episodic pattern is observed in about 10-15% of cases.

Management

Pharmacotherapy

The mainstay of OCD treatment, are the Serotonin Reuptake Inhibitors (SRIs). Until the advent of clomipramine and later the SSRIs, OCD was regarded as being a relatively refractory disorder. Studies since then have consistently shown the efficacy of SRIs over drugs acting on other neurotransmitter systems (Goodman, 2002).

Serotonin Reuptake Inhibitors

Drugs effective in OCD are also anti-depressants, but not all anti-depressants work in OCD; only those, which block the uptake of serotonin, are effective. As a matter of fact it is the therapeutic efficacy of these agents, which is the cornerstone of the serotonergic basis of OCD, which otherwise till today has not been unequivocally proven by other biological parameters. Doses of drugs are also higher in the treatment of OCD, as compared to depression, and a treatment trial is of a longer duration.

The superiority of clomipramine over placebo and non-serotonergic antidepressants has been consistently proven over time. Clomipramine was the first drug to be approved for the treatment of OCD, a little over a decade ago. Most of the side effects of clomipramine can be predicted by its receptor binding profile. The anti-cholinergic effects observed frequently include dry mouth and constipation. Sedation and weight gain are probably mediated through the anti-histaminergic profile, while alpha2-adrenergic blockade causes orthostatic hypotension. Safety concerns include prolongation of the QT interval and seizures. Like other SSRIs, sexual dysfunctions and gastro-intestinal symptoms (nausea, vomiting, diaorrhea) are mediated through the serotonergic system.

The last decade has seen the advent of SSRIs (fluvoxamine, paroxetine, sertraline, fluoxetine and citalopram) with increasing serotonergic selectivity. Most of these drugs have been found effective in treating OCD. Their usage in children has also been accepted. SSRIs are generally well-tolerated and there are few safety concerns. Based on electrophysiological evidence from laboratory animals, it has been suggested that enhancement of serotonergic transmission in the orbito-frontal cortex during chronic SRI administration is related to the anti-obsessive effect of these drugs. As SSRIs have a better tolerability profile compared to clomipramine, they are more widely used in the treatment of OCD today. However there is some evidence to suggest, from meta-analytical studies, that clomipramine offers an edge over SSRIs in the treatment of OCD, and it is fairly well established that before a patient is recognized as being treatment refractory there should have been a trial with adequate doses of clomipramine.

The first trial of an SSRI should be for 10-12 weeks. In the individual patient it may be difficult to predict the best drug, although often co-morbidity and tolerability issues will aid in the final choice of agent. In spite of considerable disability, many patients will seek therapy after years of suffering. The dose of the SSRI can be increased every 3-4 days (faster in inpatient settings) but this dose escalation should be reduced if the patient cannot tolerate the drug. Fluoxetine, paroxetine and sertraline can be given as single daily dosages, while fluvoxamine is usually preferred in divided doses. Clomipramine and fluvoxamine can be given at night because they are usually sedating; fluoxetine is usually given in the morning because it is an activating agent and can reduce sleep. Higher doses of paroxetine (40 mg +), fluoxetine (40-80 mg) and the other agents are recommended, in contrast to the practice in depression. Non-responders may respond to an escalation of dosage.

Little data is available about how long treatment should be continued in OCD. Most patients continue to take treatment for a year; most also seem to require indefinite treatment. Relapse rates with acute discontinuation are as high as 90 % in some studies. Dose reduction to a dose 40-60% of the initial dose may be a viable strategy. There is also a suggestion that gradual taper of medication over a prolonged period (e.g., 6 months or more) may also reduce the relapse rate.

If the trial of one SRI at the highest tolerated dose is ineffective, other SRIs can be tried. Before the patient can be labeled as treatment refractory, 3 trials of SRIs (including one with clomipramine) and appropriate behaviour therapy should have been used. Other agents like MAO inhibitors, venlafaxine and nefazodone have also been used.

Combination strategies which alter serotonergic function are commonly used in non-responsive patients. These include agents like clonazepam, lithium, tryptophan, pindolol, trazodone and buspirone. Another serotonin reuptake inhibitor can also be used, with a cautious observation for the occurrence of the serotonin syndrome. The use of clomipramine and an SSRI seems to offer a heuristic advantage.

Recent studies have shown the advantage of adding risperidone in the treatment of OCD. Although initially this treatment was used predominantly in the treatment of co-morbid tics, its role in independent OCD has been established in double blind trials. Specific clinical features predictive of a response to SSRI-risperidone combination have yet to be identified. An earlier study of haloperidol augmentation of fluvoxamine therapy in refractory patients reported positive results.

Other non-pharmacological strategies being investigated include Transcranial Magnetic Stimulation and Deep Brain Stimulation. ECT provides transient improvement in selected patients. Stereotactic psychosurgery is offered as a last resort by some centers.

Psychosocial intervention

Initial reports of psychoanalytical treatment of OCD were not replicated. However since the 1970s systematic studies have consistently shown the efficacy of behaviour therapy in OCD. Most practice guidelines would recommend combination of drugs and behaviour therapy, although some studies have even gone onto suggest that behaviour therapy alone would suffice in milder cases (Greist & Baer, 2002). Exposure in vitro and ritual prevention are critical elements, and gains achieved in the short term can often be maintained for long periods. However some individuals prefer not to undergo

this anxiogenic therapy, and for a few appropriate therapy cannot be devised. Patients without compulsions pose a separate challenge, and audio-exposure is an area in which more research needs to be conducted. Self help books are useful for some patients. Recent formulations have focused on cognitive errors and distortions in OCD. Cognitive behaviour therapy includes these issues along with traditional exposure and response prevention. Although the formalisation of this approach has been of recent origin, without undertaking some cognitive work, it had always been difficult to get patients to engage in exposure and response prevention tasks and maintain compliance, without addressing these issues. All the components seem to be key issues in the behavioural management of OCD. Clinical experience suggests that drug trial often produces sufficient clinical response to enable patients with OCD to engage in behaviour therapy. Often families get extremely involved in the perpetuation of rituals associated with the affected person, especially in the younger population, and especially reassurance seeking and giving assumes compulsive proportions. These issues also need to be tackled in a family setting. Often the social and occupational disability of OCD patients is so marked, that they have to be guided back into normal functioning in these areas, otherwise clinically meaningful recovery does not correlate with re-integration into the mainstream of society. Finally, while symptom amelioration is the standard with combinations of various therapies, total symptom removal is rare. Hence patients have to be taught how to live with fewer symptoms, and still lead productive and meaningful lives.

Phobic Disorders

Santvana Sharma
Shamsah Sonawalla
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Rajesh Parikh

'Are you only scared . . . ...or are you phobic?'

A 24 year old married woman presented to the psychiatric outpatient department complaining of extreme fearfulness while traveling by buses or trains over the past two months. Her symptoms commenced after her witnessing an accident involving a man falling off a running train. Subsequently she became extremely fearful whenever she had to travel. Initially, she would feel a wave of terror every morning when she would travel to go to the work place. Within a week she started avoiding the train and started taking the bus. Within a few days, she developed anxiety while traveling by buses too. The very thought of traveling would trigger episodes of severe anxiety, accompanied by trembling, sweating of the palms, feelings of suffocation, and she would feel that she would soon die. These episodes would last for 5- 10 minutes, and she would dread these intense, fearful episodes. The episodes increased in frequency, occurring up to 4-5 times a day over two months at which point she considered seeking psychiatric help. Due to these symptoms, she had stopped going to work, and even thoughts of stepping outside the house brought on the same episodes of intense anxiety. Finally, over a period of two weeks, the patient started feeling sad, was tearful most of the time, felt a sense of loss of control over her life and started wishing that she were dead. Her husband persuaded her to seek psychiatric help.

After obtaining a detailed history and conducting a thorough mental status evaluation, a diagnosis of panic disorder with agoraphobia and a comorbid diagnosis of major depressive disorder was made. The patient was started on a combination of an antidepressant, an anxiolytic and behaviour therapy. Over a period of three weeks, the patient felt considerably better and started going out of the house and eventually resumed travel by trains and buses within six weeks.

In the above case history:

1. Episodes of severe anxiety lasting for 5-10 minutes and associated with tremulousness, sweating, palpitations, chest pain and feeling that the person will die constitute a panic attack; multiple such attacks over a period of a month constitute a panic disorder. Panic disorder is also associated with a feeling of impending doom and a feeling of anxiety in anticipation of the next attack (anticipatory anxiety).

2. The patient is phobic / fearful of traveling in crowded places, in trains and buses as she fears having a panic attack and if she does travel, she experiences marked distress. This typifies agoraphobia, a type of phobia.

3. Subsequent to the panic disorder and agoraphobia, the patient has developed depressive symptoms in the form of sadness of mood, crying spells, withdrawn behaviour, feelings of hopelessness and suicidal ideation; hence the diagnosis of a comorbid major depressive disorder. Phobic disorders are associated with a high risk of comorbid psychiatric disorders, which must be recognized and treated.

4. Patient felt so fearful, and subsequently, depressed, that she stopped going to work. The impact of phobias can be enormous, leading to socio-occupational dysfunction. This is a relevant issue in differentiating normal anxiety or fear from phobias, the latter being a psychiatric disorder requiring treatment.

Phobic Disorders

Phobia is defined as 'an irrational fear that produces conscious avoidance of the feared object, activity or situation' (Ewald Horwath et al., 1998). Either the presence or the anticipation of the phobic entity elicits severe distress in an affected person who usually recognizes that the reaction is excessive. Phobic reactions usually disrupt the ability to function in life.

Phobias are the most common of all anxiety disorders. According to the Diagnostic and Statistical Manual of Psychiatry, 4th edition (DSM-IV) (American Psychiatric Association, 1994), phobias are subdivided into three categories

- Agoraphobia
- Specific phobia &
- Social phobias

Agoraphobia

The term was coined in 1871, to describe the condition of patients who were afraid to venture out alone in public places. It is derived from the Greek words 'agoro' and 'phobos'- meaning 'fear of the market place'. Agoraphobia might be defined as a fear of and an avoidance of being in places or in situations from which escape might be difficult or in which help may not be available, in the event of sudden incapacitation (DSM IV, 1994). As a result of such fears, the agoraphobic person avoids travel outside the home or requires accompaniment when away from home. Moderate cases may cause some constriction in lifestyle, while severe cases of agoraphobia may result in a person being completely housebound or unable to leave the home unaccompanied. This may be associated with panic disorder (episodic anxiety attacks with other somatic complaints, anticipatory anxiety and a feeling of impending doom) or may occur by itself i.e. agoraphobia without panic disorder.

Specific phobia

This is defined as a "marked and persistent fear that is excessive or unreasonable" and is brought on by the presence or anticipation of a specific object or situation (DSM IV). The response may take the form of a situationally bound or predisposed panic attack, and the phobia causes marked distress or interferes with role functioning. People with specific phobias, may anticipate harm, such as fear of being bitten by a dog, or may panic at the thought of losing control, for instance, if they fear being in an elevator, they may worry about fainting after the door closes.

Some common specific phobias include acrophobia: fear of heights; algophobia: dread of pain; ailurophobia: fear of cats; erythrophobia: fear of red, and panphobia: fear of several things.

Social phobia

This is defined as a strong persistent, irrational fear, accompanied by a compelling desire to avoid situations in which a person might act in a humiliating or an embarrassing manner while under the scrutiny of others (DSM IV). It is the most common type of phobia, and is further subdivided into

- Nongeneralised type-which is characterised by fear of public situations such as public speaking or performing on stage, and
- Generalized type-in which almost all interactions are feared.

Epidemiology

The life-time prevalence of agoraphobia has been reported as ranging from as low as 0.6% to as high as 6% (Ewald Howarth et al., 1998). Studies of agoraphobia in community samples have found that as many as half the patients have agoraphobia without panic disorder (Ewald Howarth et al., 1998). Women are 2-3 times more likely to be affected than men (Ewald Howarth et al., 1998). The 6-month prevalence of specific phobia is about 5-10 per 100 people. The female:male ratio is about 2:1, although the ratio is closer to 1:1 for the blood-injection-injury type (Daniel et al., 1998). The peak age of onset for the natural environment type and blood-injection-injury type is in the range of 5-9 years; although onset also occurs at older ages (Kaplan and Sadock, 1997). The peak age of onset, for the situational type (except fear of heights) is higher in the mid 20's, which is closer to the age of onset for agoraphobia (Kaplan and Sadock, 1997). The 6-month prevalence for social phobia is about 2-3 per 100 people (Ewald Howarth et al., 1998). Females are affected more often than males. The peak age of onset for social phobia is in the teens, although onset is as common at age 5 years, as it is at age 35 (Ewald Howarth et al., 1998).

Etiology

Behavioural factors

Watsons hypothesis invoked the traditional Pavlovian stimulus-response model of the conditioned reflex to account for the creation of the phobia. Anxiety is aroused by a naturally frightening stimulus that occurs in relation with another neutral stimulus. As a result of the association, especially when the two stimuli are paired on several successive occasions, the originally neutral stimulus takes on the capacity to arouse anxiety by itself. The neutral stimulus therefore becomes a conditioned stimulus for anxiety production. Also, certain actions enable the person to avoid the anxiety-provoking object or situation. Such avoidance behaviour becomes fixed as a stable symptom because of its effectiveness in protecting the person from phobic anxiety. In social phobia, several studies have reported that some children possibly have a trait characterised by a specific pattern of behavioural inhibition. It is also observed that parents of people with social phobia are less caring, more rejecting and more overprotective of their children, compared to other parents.

Psychoanalytic factors

Freud viewed the phobia-anxiety hysteria as a result of conflicts catered on an unresolved childhood Oedipal situation (i.e. fixated at the phallic stage of Freuds psychosexual stages of development); when repression fails to be entirely successful, the ego must call on auxiliary defences.

In patients with phobias, the defence mechanism primarily involved includes the use of displacement, that is, the sexual conflict is displaced from the person who evokes the conflict to a seemingly unimportant, irrelevant object or situation, which then has the power to arouse a constellation of affects, including signal anxiety. The phobic object or situation may have a direct

associative connection with the primary source of the conflict, and thus symbolizes it (that is the defence mechanism of symbolization). The situation or the object is usually one that the person can keep away from, with the additional defence mechanism of avoidance, thereby escaping suffering from severe anxiety. Counterphobic attitude-phobic anxiety can be hidden behind attitudes and behaviour patterns that represent a denial. In this situation a person reverses the situation and actively attempts to confront and master whatever is feared e.g. parachute jumping, rock climbing may exhibit counter phobic behaviour. Other mechanisms of associations between phobic object and the phobic emotions include modelling in which the person observes the reaction in another (for example, a parent) and information transfer, in which a person is taught or warned about the dangers of specific objects (e.g. venomous snakes).

Biological factors

Neurochemical factors

There is an adrenergic hypothesis for phobias, especially social phobias (Sullivan et al., 1998). Patients with performance phobias may release more norepinephrine or epinephrine, both centrally and peripherally, than do nonphobic people, or such patients may be sensitive to a normal level of adrenergic stimulation. Also, some investigators have hypothesized that dopaminergic activity is related to the pathogenesis of this disorder (Charney DS et al. 1998; Sullivan et al., 1998).

Genetic factors

First degree relatives of people with social phobia are about three times more likely to be affected with social phobia than are the first degree relatives of those without mental disorders (Ewald Horwath et al., 1998). Specific phobias tend to run in families (Abby Fyer, 1998). The blood-injection injury type has a particularly high familial tendency. Studies have reported that two-thirds to three-fourths of affected probands have at least one first -degree relative with specific phobia of the same type (Abby Fyer, 1998).

Clinical features

Patients with agoraphobia avoid situations in which it would be difficult to obtain help. They prefer to be accompanied by a friend or a family member in busy streets, crowded stores, enclosed spaces (such as tunnels, bridges and elevators), and enclosed vehicles (such as subways, buses and airplanes). The patients may insist that they be accompanied by someone every time they leave the house. Severely affected patients may simply refuse to leave the house. Other co-morbid disorders associated with agoraphobia are major depressive disorder, panic disorders obsessive-compulsive disorder and other phobias. Secondarily, it can lead to marital discord, time lost from work, financial difficulties related to the loss of work, alcohol and other substance use.

Various types of specific phobias have been described i.e. irrational fear of specific objects or situations. These are: animal type, natural environment type (for example storms), blood-injection injury type, situational type (for example cars, and other types (for specific phobias that do not fit into the previous four types). Patients have severe anxiety when made to face a specific object or situation, hence leading to avoidance of that object/situation, for example, a patient may take a bus,

rather than fly, across long distances, to avoid contact with the object of the patients phobia, i.e. an airplane.

In social phobia, there is marked avoidance of social or performance situations in which there is exposure to unfamiliar people or to possible scrutiny by others. The individual fears that he or she will act in a way (or show anxiety symptoms) that will be humiliating or embarassing. This avoidance, anticipation, or distress interferes significantly with the person's normal routine, occupational functionig, or social activities. It may be generalised when the fears include most social situations.

Differential diagnosis and Comorbidity

Phobias need to be differentiated from appropriate fear and normal shyness. For a diagnosis of phobic disorders, the symptoms must be noted to impair the patient's ability to function appropriately. Nonpsychiatric medical conditions that can result in the development of phobia include the use of substances (particularly hallucinogens and sympathomimetics), central nervous system tumours, and cerebrovascular disorders. Phobic symptoms in these instances are unlikely in the absence of additional suggestive findings on physical, neurological and mental status examinations.

Patients with schizophrenia can have phobic symptoms as part of their psychosis. Unlike patients with schizophrenia, patients with phobia have insight into their illness. Panic Disorders, agoraphobia, and avoidant personality disorder also must be considered. In general, patients with specific phobia or non-generalized social phobia tend to experience anxiety immediately when presented with the phobic stimulus, and the anxiety is limited to the identified situation. Patients with agoraphobia are often comforted by the presence of other people in an anxiety-provoking situation, whereas patients with social phobia become more anxious in the presence of people. Panic disorder and agoraphobia are associated with breathlessness, dizziness, a sense of suffocation and a fear of dying, whereas in social phobia, symptoms include blushing, muscle twitching and anxiety regarding scrutiny from others. Avoidant personality disorder is a pervasive pattern of behaviour where a person avoids people and situations. It may be difficult to differentiate from social phobia but requires extensive interviews and detailed psychiatric history. Other differential diagnoses to be considered include schizoid personality disorder and major depressive disorder. Patients with major depression are likely to present with the constellation of depressive symptoms. In patients with schizoid personality disorder, there is lack of interest in socializing, rather than fear of socializing, which leads to the avoidant social behaviour. Hypochondriasis, obsessive compulsive personality disorder, and paranoid personality disorder need to be considered in the differential diagnosis of specific phobia.

Depression may complicate the symptom picture in 40-80% of patients with agoraphobia and is also strongly associated with specific and social phobia. These patients are at an increased risk for suicide. Alcohol and other substance dependence disorders occur in about 20-40 % of patients, and obsessive-compulsive disorder may develop. Family interactions, performance in school and at work commonly suffer.

Course and prognosis

Agoraphobia

Most cases of agoraphobia are associated with panic disorder; when the panic disorder is treated, the agoraphobia often improves with time. However, agoraphobia may also occur by itself and requires behaviour therapy. Agoraphobia without a history of panic disorder is often incapacitating and chronic, and depressive disorder and alcohol dependence often complicate its course.

Specific phobia and social phobia

Patients with specific phobia and social phobia may be dependent on others and may have significant impairment in their social and occupational functioning; in the case of young persons, school performance may be impaired (Kaplan and Sadock, 1997). The associated substance-related disorders can also adversely affect the course and prognosis of the disorders. However, the literature on the course and prognosis of specific and social phobias is not very detailed at present.

Management

In patients with agoraphobia with panic disorder, and in patients with social phobia, the most effective treatments include a combination of pharmacotherapy and behaviour therapy. In specific phobia, behaviour therapy is predominantly used, although adjunctive pharmacotherapy may be used (Laszlo A et al., 1998).

Pharmacotherapy

1. Benzodiazepines (BDZ)- These have the most rapid onset of action against panic, often within the first week. Alprazolam has been the most widely used, but even lorazepam and clonazepam have shown efficacy. Some patients use these drugs when faced with the phobic stimulus. Benzodiazepines are not generally used for a long period because of their addictive potential, cognitive impairment and potential for abuse. Although benzodiazepines may be used initially, simultaneous treatment with an antidepressant (e.g. an SSR- serotonin specificre uptake inhibitor) must be initiated; after a period of 8-12 weeks, the BDZ can be gradually tapered and stopped and the SSRI continued.

2. Serotonin- Specific Reuptake Inhibitors (SSRIs)- Paroxetine and sertraline have been approved for the treatment of panic disorder with co-morbid agoraphobia. Fluoxetine, fluvoxamine and citalopram may also be used, especially when co-morbid with major depressive disorder.

3. Serotonin- Norepinephrine Reuptake Inhibitors (SNRIs)- Venlafaxine has shown some benefit in patients with social phobias and may be used.

4. Buspirone- There is some evidence of its efficacy in patients with social phobia, especially when used to augment treatment with SSRIs. However the therapeutic doses are achieved after 8-12 weeks, hence can be used along with a faster acting drug.

5. Tricyclics and Tetracyclics- Clomipramine and imipramine have shown the most benefit in patients with panic disorder co-morbid with agoraphobia, but the benefit may not be achieved until

the adequate therapeutic dosage is reached and may require 8-12 weeks. Also, these group of drugs are now used less frequently compared to the SSRIS in view of the more serious side effects at the higher dosages required for effective treatment of Panic Disorder. There is some evidence for efficacy of desipramine and somewhat less evidence for the efficacy of maprotiline, trazodone, nortryptiline, amitryptiline and doxepin.

6. MAO Inhibitors (MAOIs)- Phenelzine and tranylcypromine have shown some efficacy, but require full dosages of 8-12 weeks to be effective. Reversible inhibitors of MAO, such as moclobomide and brofaromine have shown some efficacy in patients with social phobias.However the role of dietary restrictions has limited the use of MAOIs, particularly since the appearance of SSRIs.

7. Betablockers- Atenolol and propanolol have shown efficacy when administered shortly before exposure to a phobic stimulus in patients with social phobia associated with performance situations and in patients of specific phobia.

Psychosocial interventions

a) Insight oriented Psychotherapy- This focuses on helping patients understand the hypothesized unconscious meaning of the anxiety, the symbolism of the avoided situation, the need to repress impulses, and the secondary gains of symptoms.

b) Supportive Psychotherapy- This is useful in helping the patient to actively confront the phobic object during treatment.

c) Behaviour therapy- Behavioural techniques like relaxation and breathing control can be used to desensitize patients using a series of gradual self-paced exposures. Techniques include exposure therapy, systematic desensitisation, graded exposure, flooding and implosion.

d) Cognitive behaviour therapy- This includes behavioural techniques described above and cognitive approaches. The cognitive approach reinforces the realisation that the phobic situation is, in fact, safe.

e) Hypnosis- This technique is used to enhance the therapists suggestions that the phobic object is not dangerous, and self hypnosis can be taught as a method of relaxation when confronted with the phobic object.

f) Family therapy- This can enlist the families aid in treating the patient but it may also help the family understand the nature of the patients problem.

The most commonly used treatment for specific phobia is exposure therapy, a type of behaviour therapy in which the therapist desensitises the patient using a series of gradual, self paced exposures to the phobic stimulus. The patient is also taught various techniques to deal with the anxiety, including relaxation, breathing control and cognitive approaches. Psychotherapy for the generalized type of social phobia involves a combination of behavioural and cognitive methods, including cognitive retraining, desensitization, rehersal during sessions and a range of homework assignments. Insight-oriented psychotherapy may benefit patients with agoraphobia, specific and social phobias. Hypnosis, family therapy and supportive therapy may be useful in treating phobias.

Conclusions

It is important to differentiate normal fear and anxiety from phobias due to comorbid psychopathology, which may adversely affect socio-occupational functioning. Hence the necessity for early diagnosis and treatment, which may help prevent psychiatric morbidity. A combination of pharmacotherapy and psychotherapy is reported to give the best results. At present, there is a wide choice of drugs to choose from and various psychotherapies, which can be used to improve the outcome for our patients who are 'trapped in their own fears'.

Posttraumatic Stress Disorder

Harish Shetty

'Case of re-experiencing accident'

Mr. Bhagirath, aged 32 years, presented with the symptom of difficulty in falling asleep for the last three months. He had been consuming Tablet alprazolam 0.25 mg once or twice a day with little benefit. According to him, the symptoms began after a car accident in which he was involved. He was on his way back from a religious place with his family, when a speeding truck hit his car. They were miraculously saved without a scratch, but the car was badly damaged. Since then, he has not been able to sleep well and would get up with a startle when disturbed by a small noise. He experienced the same during the day when noise of a truck or other similar noises would startle him and disrupt his routine work. He found it difficult to concentrate and was disturbed by the thoughts of the accident. At times, he would feel that the accident was actually happening in the present and would behave as if he was in the car. Mr. Bhagirath tried his best to avoid the thoughts and push it away. He also avoided the road on which he had the accident and was scared to travel in cars. He never had such symptoms in the past. He also complained of easy irritability and memory problems such as difficulty in recalling the notes read on the previous day.

Bhagirath is suffering from Posttraumatic stress disorder as he is experiencing a group of symptoms which have persisted even after one month of the actual occurrence of the life threatening accident. These group of symptoms include, recurrent thoughts about the accident, flashbacks i.e., re-experiencing the accident, avoiding roads and cars, startle reaction on hearing the noise of trucks, insomnia, easy irritability, difficulty in concentration and memory problems.

Posttraumatic Stress Disorder (PTSD)

After exposure to a traumatic life threatening accident or natural disasters like earthquake, cyclone, floods or man-made disasters like bomb blasts and riots, most of the people involved in it or witnessing it develop a group of symptoms termed as acute stress reaction. These symptoms usually resolve gradually over a period of one month. In some susceptible individuals these symptoms persist beyond one month and cause severe distress and functional impairment. These patients are then diagnosed as suffering from PTSD.

Epidemiology

Life time prevalence rates of PTSD ranges from 5-6% in males and 10-12% in females (Kessler et al., 1995; Breslau et al., 1991).

Etiology

Various neurotransmitters like noradrenaline, serotonin, and dopamine have been implicated in the pathogenesis of PTSD (Southwick, 1997). Opioid system and Hypothalamic-Pituitary-Adrenal Axis also play a significant role in the etiology of PTSD.

Clinical features

Following clinical features may suggest the diagnosis of PTSD.

- The person should have experienced or witnessed or confronted with an event or events that

involved actual or threatened death or serious injury, or a threat to the physical integrity of self or others

- The person's response should include helplessness, fear or horror
- Recurrent intrusive thoughts
- Recurrent distressing dreams of the incident
- Acting out as if the event is actually happening in the present
- Intense psychological distress when confronted by cues which symbolizes or resembles the incident
- They may also have symptoms of avoidance evident by the following symptoms
  1. Efforts to avoid thoughts, feelings, or conversations associated with the trauma
  2. Efforts to avoid activities, places, or people that arouse recollections
  3. Inability to recall an important aspect of the trauma
  4. Markedly diminished interest or participation to significant activities
  5. Feeling of detachment or estrangement from others
  6. Sense of foreshortened future (e.g., does not expect to have a career, marriage, children, or a normal life span)

- Increased arousal as indicated by following symptoms is also commonly seen in these patients
  1. Difficulty falling or staying asleep
  2. Irritability or outbursts of anger
  3. Difficulty concentrating
  4. Hypervigilance
  5. Exaggerated startle respons

Some survivors may rise to the occasion and help others effectively during the recovery phase. Such patients may have a delayed onset of symptoms which one needs to be aware of.

Some of the individuals exposed to traumatic event may try to cope up with this situation by indulging in excessive religious activities (excessive praying, going on pilgrimage and building expensive places of worship or donating big amounts for the same) or by consuming addictive substances like alcohol, cannabis and opium derivatives.

Course and Prognosis

The full syndrome of PTSD exhibits a variable course with some evidence that, this also relates to the nature of the trauma. A large majority of patients experience complete remission, while others exhibit only mild symptoms. Approximately 10 percent of patients with PTSD exhibit a persistent or chronic course.

Management

Prevention of PTSD

Implementation of following steps immediately after the disaster may prevent the PTSD in the exposed individual.

- Provide basic amenities like food, clothing and shelter.
- Allow the survivor to perform the rites and rituals for the family members or other community members who have lost their lives in the disaster at an appropriate time.
- Encourage the patient to express and ventilate his feelings associated with the traumatic event without being critical or judgemental.
- Discourage the patient to get involved in superstitious practices and do not instigate the patient to demand unreasonable compensations.
- Encourage him to resume his daily chores and occupational activities at the earliest.

Management of PTSD

- Avoid indiscriminate use of benzodiazepenes. Use of this group of medications should be avoided as it has a strong potential for dependence. Addiction following disasters is well-known.
- Do not advise change of residence or avoidance of the places/objects/situations reminding of the disaster, to diminish the symptoms.
- Do not advise the patient to, ' forget about the disaster and start thinking positively'.
- Do not advice the patient to rely excessively on prayers and God.
- Do not give long discourses asking the patient to improve his will power and fight the symptoms.

Referral to a psychiatrist

If PTSD patient exhibits excessive agitation, is suicidal or homicidal or develops alcohol abuse or dependence then it is preferable to refer him to a psychiatrist.

Pharmacotherapy

Antidepressants like amitriptyline (50-150 mg/day), imipramine (50-150 mg/day), fluoxetine (20-60 mg/day), sertraline (50-200 mg/day), paroxetine (20-60 mg/day), fluvoxamine (50-150 mg/day), citalopram (20-60 mg/day) have been effectively used in the treatment of PTSD and associated anxiety and depression.

These medicines once initiated may have to be continued for a period of 6-8 months. Once the patient has completely recovered it may be gradually tapered and discontinued. Abrupt discontinuation of antidepressants may cause withdrawal symptoms.

Psychosocial intervention

Different types of psychotherapies like supportive psychotherapy, cognitive behavior therapy, relaxation therapy, systematic desensitization and eye movement desensitization and reprocessing therapy have been tried.

Conclusion

PTSD is a preventable and treatable psychiatric disorder. Early intervention, appropriate and judicious use of antidepressants, along with psychosocial intervention, helps in the reduction of distress and leads to substantial recovery and improvement in the quality of life.

Anxiety Disorders and Depression in Children and Adolescents

Nilesh Shah
Om Prakash Raichandani

'Refusal to School' A Case Report

Raju, a 4 year old boy was brought by his parents to the Child Guidance Clinic with history of refusing to attend school. According to his parents, about 3 months back, he was put in a nursery school. When his mother took him to school on the first day, like many other children of his class, he was reluctant to separate from his mother and refused to enter the class. When he was persuaded by his class-teacher, he demanded that his mother should sit with him in the class. As he was very adamant, the teacher allowed his mother to be with him in the class. Over the next couple of weeks, his mother noticed that though, all the other children were coming to school very willingly and attending the class by themselves, Raju persisted with his demands. He would not sit in the class without his mother and if separated forcibly, he would keep on crying and refuse to participate in any of the activities. After a few days, he started refusing to go to school. Every morning, as soon as his mother would dress him up in his school uniform, he would start crying and express his unwillingness to go to school. In spite of his refusal, when the mother remained firm and took him to school, he would keep on crying on the way and would not sit in the class without his mother. When even after 3 month of persuasion, bribes, rewards, scolding, punishments etc., his refusal to school continued, he was brought to the Child Guidance Clinic by his parents.

On further enquiry, it was also gathered that it was not only in school, but also in other situations, Raju avoided separation from his parents. He would not go out of his house to play with other children alone or would not go to the garden with his cousins, uncles or neighbors. He would also insist on sleeping with his parents at night and in spite of persistent efforts his parents did not succeed in convincing him to sleep by himself in his room.

As evident in the above case report, Raju has developmentally inappropriate behavior when separated from his parents. He is reluctant to go to school and refuses to sit alone in the class. He shows similar behavior in other situations as well, like refusing to go out with his cousins and insisting on sleeping with his parents. Thus, he seems to be suffering from separation anxiety disorder.

Separation Anxiety Disorder

In this disorder, the child is reluctant to separate from his parents or a close familiar person and the intensity of the reaction to separation is out of keeping with his developmental age. It is common in children in the age group of 3-6 years. It occurs in boys as well as girls. The estimated prevalence of this disorder is around 3 to 4 percent of all school-age children. The most well recognized expression of this disorder is refusal to go to school and so it is also known as school phobia (Last and Strauss, 1990). If the child is forcibly separated and sent to school he may develop various somatic symptoms like pain in abdomen, vomiting or headache.

It is important to recognize this disorder and make parents and teachers aware about this disorder. Initially it may be a good idea to allow one of the parents to sit with the child in the class. Once the child develops the trust that the mother is not going to leave him alone in the class and go away, (which may sometime take a couple of weeks to a couple of months), she may sit outside the classroom and child may be allowed to go out and meet her as and when he wants. Gradually then, the mother may go out

of the school for a while (for a few minutes at a time) after taking the child into confidence. Finally, once the child is found to be ready for separation, the mother may go home after leaving him in school and may come back to pick him up when the school is over. The whole process of separation should be carried out very patiently and in a very non-threatening manner as it may take a couple of months or more.

Anxiety symptoms in children and adolescents

Symptoms of anxiety are not very unusual in children and adolescents. Children can develop a variety of anxiety disorders, which are seen in adults such as generalized anxiety disorder, panic disorder, obsessive compulsive disorder, phobic disorder, posttraumatic stress disorder etc. But apart from these disorders, there are certain anxiety disorders, which are specifically seen in children and adolescents such as separation anxiety disorder (Bernstein et al, 1996).

The rate of generalized anxiety disorder in school-age children is estimated to be around 3 percent, the rate of social phobia is 1 percent, and the rate of simple phobia is 2.4 percent (Kaplan & Sadock, 2001).

The experience of anxiety has two components, the awareness of the physical sensations, (such as palpitations and sweating), and the awareness psychological sensations, of being nervous and frightened. The experience of severe anxiety affects thinking, perception and learning.

In younger children, the symptoms of anxiety are more psychological and manifest in the form of unusual behavior like refusing to separate from parents or avoidance of new interpersonal contacts. In older children and adolescents, the symptoms of anxiety may be more at cognitive level like excessive concern and worries.

The other two anxiety disorders in children and adolescents described in the former classification (DSM-III) (American Psychiatric Association, 1980) along with the separation anxiety disorders were `avoidant disorder' and `overanxious disorder'. The children having these disorders may now be diagnosed as `Social Phobia' or `Social Anxiety Disorder of Childhood' and `Generalized Anxiety Disorder' respectively.

'Mama's boy' A Case Report

Vikas refused to enter the Child Guidance Clinic even though he saw many toys and games inside, which he was very fond of. After some persuasion he entered the clinic clinging to his mother, hiding behind her. He did not make any attempt to separate from his mother and play with other children in the clinic. His mother was feeling very upset and embarrassed with his behavior. According to her, Vikas has been very shy, doesn't make friends and prefers to remain aloof even at the age of 7 years. Even in the school he hardly talks or plays with anyone. He does not initiate any communication and does not actively participate in any activities. Though, at home or in a familiar environment and with only a couple of familiar persons he is friendly, in most of the other situations, he doesn't mix or socialize. When they have guests, he doesn't greet them and remains in his room. He is described as a very shy, timid and mama's boy in the family.

Vikas has Avoidant Disorder or Social Anxiety Disorder of childhood.

Avoidant Disorder

Children or adolescents suffering from avoidant disorder have a persistent age inappropriate anxiety for strangers. They are very shy, remain aloof, and avoid social encounters. This disorder seems to be more frequent in girls. It is also known as social anxiety disorder of childhood (ICD-10) (World Health Organization, 1992).

Adults having excessive concern about their attire, manners and behavior in a variety of social situations that require interaction with people and who avoid social encounters may be diagnosed as social phobia (Francis, et al, 1992) or avoidant personality disorder (DSM-IV) (American Psychiatric Association, 1994) or anxious (avoidant) personality disorder (ICD-10) (World Health Organization, 1992).

Typically children having avoidant disorder do not show any embarrassment and behave with adequate amount of boldness in their own homes and with those with whom they are familiar. They may also exhibit clinging and overly demanding behavior at home. But in the social situations they try to remain inconspicuous, often talking in whispers and hiding behind people. They find it difficult to form any sort of relationship with the opposite sex and shrink from participation in competitive activities. They do not take the initiative or lead in any activity in spite of having an intense desire to do so. They may become tearful and anxious when forced into social situations. Children with this disorder may not reach their educational potential unless they are given support. Sometimes, in contrast to their outward behavior, they may be harboring very grandiose fantasies and high aspirations.

The treatment of this disorder is directed to help the child to increase assertiveness in social situations. Appreciations and encouragement aid in the process of developing assertiveness. These children should not be forced to socialize but should be allowed to take their own time to feel comfortable and socialize. Antianxiety and antidepressant drugs may be used when indicated (Birmaher et al, 1994).

'Exam Phobia' - A Case Report

Geeta was brought to Child Guidance Clinic by her parents as she used to get very anxious at the time of exams. She was 12 year old and was studying in 5th standard. According to her parents She was very much afraid of appearing in any exams. Few days before any exams, whether it is unit-test, mid-term or annual exam, she would have difficulty in falling asleep. She would start worrying about her performance and would get preoccupied' with what will happen if she gets less marks or fails'. As a result, she was unable to concentrate on her studies. She would remain restless, upset and would get irritated over trivial matters during exam times. Once the exams were over she would feel relieved. On enquiry, it was noticed that apart from exams, she used to have excessive anxiety in other situations as well. If she had to go out alone for a picnic or to a friend's house, she would be anxious and worried about a variety of things which could possibly happen over there. Frequently, she would avoid participating in extracurricular activities as she would get very anxious even at the thought of it. She would need lots of reassurance and encouragement all the time.

As evident from the above case report, Geeta has a tendency to develop excessive anxiety and worry in a variety of situations, which interferes with her performance. She is suffering from Overanxious Disorder or Generalized Anxiety Disorder.

Overanxious Disorder

There are some children and adolescents who have an excessive tendency to worry. They worry about a variety of things such as examinations, potential injuries, friendships and group acceptance. They are overburdened with ruminative concern about competence and performance. These children have perfectionist standards. They are dependent on the evaluation of others and feel rejected easily. They are usually described as very obedient and conforming children. Anxiety symptoms, difficulty in falling asleep and various somatic symptoms like headache, sore throat, difficulty in breathing are common in these children as they have anticipatory anxiety about forthcoming events.

Adults having similar excessive anxiety and worry about a number of events or activities may receive the diagnosis of generalized anxiety disorder. In children and adolescents, instead of the term `overanxious disorder', one may prefer to use the term `generalized anxiety disorder' or `generalized anxiety disorder of childhood'.

Individual psychotherapy and judicious use of antianxiety medications have been found to be very useful in this disorder (Kranzler, 1988). Due to great motivation to please, high level of understanding ability, these children usually do exceedingly well in psychotherapy. Antianxiety drugs may be particularly very useful when there are physical symptoms of anxiety and insomnia.

Childhood Depression - A Case Report

Rita, a 4 year old girl was brought to the Child Guidance Clinic by her mother as she had observed a remarkable change in her behavior in the past 3 to 4 weeks, prior to which she was fine - going regularly to the school, playing with her brother, demanding toys, clothes, chocolates etc. The mother had noticed that, without any obvious precipitating factor, in the past 4 weeks, she had gradually turned irritable and cranky. In the morning, she would be reluctant to go to school. In the school, teacher had also noticed that she was remaining very quiet and aloof. She would not be very willing to participate in group activities and would start crying over trivial provocation by her class-mates. In the evening at home, she would keep clinging to her mother and would not allow her to go out anywhere and would insist that she should stay with her. She would get very frightened and start crying if there was any kind of noise. She had stopped demanding anything and would not play with her brother or friends. On further enquiry, the mother recalled that she had a similar episode about 2 years back during the same time of the year, which had lasted for about a month and had improved completely by itself without any treatment.

Rita seems to be having a second episode of depression. She has persistent sadness of mood and irritability for more than 2 weeks, which has affected her performance in school and overall behavior at home. In medical terminology, this would be called as `Major Depressive Disorder Recurrent' or 'Recurrent Depressive Disorder'.

The other two disorders in which depressive symptoms are observed in children are `Dysthymic Disorder' and `Adjustment Disorder with Depressed Mood'. Dythymic disorder is a chronic disorder characterized by presence of depressed or irritable mood with other symptoms of depression lasting for more than a year. Previously, it was referred to as `Neurotic Depression'. On the other hand, when

minor (less severe and short lasting) depressive episode follow a significant stressful life event like failure in exams or love affair it is diagnosed as `Adjustment Disorder with Depressed Mood' (reactive depression).

Though not very common, children do develop depression. The prevalence rate has been estimated to be around 0.3% to 2% in school going children and around 5% in adolescents. Some clinic-based studies have reported depression more common in boys than in girls in school-age children.

Children or adolescents who develop depression may be genetically predisposed to develop this disorder. Various studies in depressed children have revealed biological abnormalities similar to that found in adults. Some studies have implicated psychosocial factors like loss of parents in early childhood, for development of depression (Birmaher et al, 1996; Harrington, 1990; Kazdin, 1990).

As in case of Rita, it may start spontaneously or after a minor precipitating event. Children and adolescents may be more irritable rather than depressed during this period. Younger children may show excessive clinging to their parents and may turn cranky. As studies and play are the main occupations of the children, impairment in these two areas is prominent. In some children, loss of appetite and weight or failure to gain weight may be a prominent feature. Adolescents may indulge in substance use, antisocial activities, and impulsive behavior like running away from home or attempting deliberate self harm during the phase of depression.

Antidepressants like imipramine and amitriptyline have been used in the treatment of depression in children, but recently newer antidepressants like fluoxetine or sertraline are more preferred as they are better tolerated and are relatively safe in overdose. Though very effective and safe, ECT is rarely used in children and adolescents. Its use may be life-saving in severe, treatment-resistant depression associated with psychosis, catatonic symptoms or persistent suicidality.

Anxiety and Depressive Disorders in Women

Prabha S.Chandra

Introduction

Epidemiological and clinical studies have consistently documented that depression across different cultures is about twice as common among women than men. Women in the reproductive age group are subjected to unique neuroendocrine alterations. In addition, the psychological status and role of women in society, place them in a vulnerable position in times of stress. The interaction of these factors in the pathogenesis and maintenance of depression necessitates special consideration in evaluation and management of women with anxiety and depressive disorders.

This chapter describes postpartum depression and premenstrual syndrome; two of the most commonly encountered psychiatric conditions in women.

Case of Postpartum depression

Mrs. Sarita, a 28 year old married woman, presented to the clinic with feelings of sadness, crying spells, decreased interaction with family members and decreased sleep and appetite. She had delivered a female baby a month earlier and the immediate postpartum period had been uneventful. She had been looking forward to the baby, however after four weeks of the birth, she reported losing interest in the infant and did not feel like nursing her and would also get irritable when the baby cried. She would feel very guilty about this and started avoiding the baby. She even complained of lack of appetite, feelings of tiredness and there was considerable weight loss. Despite the fact that she did not feel like caring for the baby, she reported excessive worrying and thoughts that something might happen to her child when someone took the baby away. There was no earlier history of depression. Her mother reported a similar history in Sarita's elder sister who also had depression in the postpartum period.

This is a case of Post-partum depression.

The important features in this case are elaborated below

1. Onset, four weeks following delivery
2. Depression, that was severe enough to interfere with routine functions
3. Neglect of infant
4. Anxieties and worries related to the infant
5. Family history of postpartum depression in sister

Postpartum depression

'Postpartum blues' refers to a brief period of dysphoria, which occurs during the first week after delivery, in about 50% of recently delivered mothers. Its symptoms include depressed mood, crying spells, irritability, anxiety, mood lability, confusion, sleep and appetite disturbances, headache, depersonalisation and forgetfulness ranging from a few hours to a few days. 'Postpartum depression' refers to those nonpsychotic mood disorders that meet the criteria for major depression.

There is a lack of consensus on the definition of the temporal aspects of the postpartum period (Cooper et al., 1995). Some consider, only the first two to three weeks as the true postpartum period,

while others suggest a period up to one year. However, most of the researchers consider three to six weeks as an appropriate time period with a maximum limit of six months.

Etiology

Postpartum depression is not a single entity, as it is often dealt with, but it is a rubric for a wide variety of disorders.

Biological factors

The biological factors implicated in the cause of postpartum depression include genetic and endocrine factors.

Genetic predisposition

Several studies have found a positive family history among postpartum depressed patients. Many women with postpartum depression have a history of previous depression.

Endocrine theory

Both indirect and direct evidence provide support for the hormonal basis of postpartum depression. The indirect evidence, consists of an association between menstrual difficulties and postpartum mood alterations.

The most commonly implicated endocrine cause for postpartum depression is thyroid dysfunction, more specifically hypothyroidism. Thyroid disturbances are mainly implicated in late onset postpartum depression. 10% of women have postpartum hypothyroidism, peaking at four to five months postpartum. Fluctuations in the oestrogen, progesterone, cortisol, tryptophan and catecholamine levels also have been implicated in the causation of postpartum depression. However, there is little consistency in the studies of hormonal variables.

Psychosocial factors

Postpartum depression is associated with many adverse psychosocial factors. They can be discussed under the following headings.

Gynaecologic and Obstetric factors

For depression, there is no consistent association with parity. Early reports of an association with physical complications or a bad obstetric history have not been confirmed. In fact, a few studies reported that these conditions decrease the frequency of postpartum depression, as women with a bad obstetric history receive special care. There have been, however, some reports of an association with caesarean section, and previous miscarriage. There is no consistent evidence regarding breast feeding or bottle feeding and postpartum depression. Some studies have reported an association between depression and having a female child while others have found no consistent relationship.

Personality and Attitudinal factors

Frequent correlation of postpartum mood changes with severity of anxiety during pregnancy has been reported. Negative maternal attitudes towards child rearing and family have been associated

with mainly anxiety and depression in the postpartum period. Studies have also shown that an unplanned pregnancy may result in ambivalence towards the child antenatally, difficulties in adjusting to parenthood and feelings of entrapment all of which lead to postpartum depression.

Life event factors

There is increasing evidence suggesting that stressful life events are a significant factor in the development of depression. Since childbirth is a significant stressor in its own right, additional stressors occurring during the course of pregnancy or in the early postpartum period may play a role in the causation of postpartum depression.

Social support factors

Studies have shown that social support has a significant impact on the stress-illness equation, either as a mediating or as an independent variable. Social support, would appear to be all the more relevant for postpartum women, who, because of the increased demands imposed by the birth of a child, may require additional emotional and instrumental support. Although some studies have implicated a lack of support from persons other than the spouse in the onset of postpartum depression, results of majority of investigations point more specifically to marital conflict and a lack of spousal support. However, certain issues remain unresolved, e.g. does depression influence the perception of social support? or does decreased quality of social support increase the vulnerability to depression?

Course and Prognosis

Postpartum blues

Postpartum blues is a transient, self limiting, emotional reaction which is specific to the childbirth. It usually remits without any treatment, within a week to 10 days. The major negative consequences of the blues, is that they may herald a more serious postpartum depression later.

Postpartum depression

Postpartum depression has a good prognosis with early detection and treatment, although its onset may be more insidious and not appear until the third or fourth month postpartum. Usually improvement is observed within 36 months, but some may develop chronic and refractory depression extending up to 4 years postpartum. The important poor prognostic factors, include patients with severe postpartum blues, high prenatal anxiety, past history of postpartum or nonpostpartum depression, antepartum depression, adverse psychosocial factors and presence of pregnancy and labour related complications.

The risk of subsequent postpartum depression (PPD) is around 20-30% compared to the risk of subsequent nonpostpartum depression (NPPD) which is around 30-50%. Patients who have only postpartum depression have around 40% risk of future postpartum depression, and 35% risk of nonpostpartum depression. Patients with both postpartum and nonpostpartum depression have 18% risk of further PPD and 60-65% risk of NPPD. The consensus view is that patients who have only postpartum depression have better prognosis, tend to relapse less and have less mean duration of total hospitalisation (Cox et al., 1993).

Management

Screening

The psychiatric history of a new obstetric patient should be routinely obtained. Prospective mothers should be assessed for any previous treatment for psychological or emotional problems. Given the high risk of relapse after delivery for these women, intensive monitoring for at least 30 days and then less intensive monitoring for an additional 60 days is appropriate in the postpartum period.

Regarding postnatal assessment, every patient should be inquired about her mood before sending her home from the hospital. With respect to patients with past history of postpartum depression, it is useful to administer appropriate screening tools such as Edinburgh postnatal depression scale (EPDS) or the Beck Depression Inventory (BDI) (Cox et al., 1987).

An important principle in screening postpartum psychiatric disorders is the identification and treatment of biological risk factors. Thyroid dysfunction is one such factor. Social risk factors are also important and need to be identified in order to provide additional support.

Another important principle guiding treatment is early detection and prompt treatment.

Assessment in Postpartum Depressive Disorders

- Past history of depression including postpartum episodes
- Marital problems (a strong predictor of PPD)
- Family history of depression
- Past or current history of thyroid dysfunction
- Assessment of the severity of the episode
- Assessment of mother infant interactions
- Assessment of any risk to the infant from the mother

When to suspect postpartum depression?

- When a mother is socially withdrawn
- When infant care is inadequate
- When there is lack of interest in any activity
- When there is a lack of pleasure in any activity
- When a mother appears dull and quiet
- When she is unduly irritable
- When she cries easily
- When she expresses over concern about the infant

Pharmacotherapy

Before embarking on drug treatment of postpartum psychiatric disorders three issues need to be considered

1. Presence of organicity, such as hypothyroidism, cortical venous thrombosis or pelvic infection
2. Safety of drug in lactating women
3. The need for prophylaxis (safety of drug during pregnancy)

Postpartum blues do not usually need any pharmacological intervention. Reassurance, advice, suggestion is all that is necessary.

Postpartum depression should be treated with either tricyclic antidepressants or SSRIs. The sideeffect profile and their sedative properties can decide the particular choice of these antidepressants. ECT should be considered in severely suicidal, homicidal, agitated, depressive women.

Use of antidepressants in pregnant women

In women, with histories of mild to moderate single episodes of depression who become pregnant or are taking antidepressants and wish to conceive, a trial of drug tapering or discontinuation is appropriate. The use of cognitive behaviour therapy may be a possible alternative in some patients.

If the patient has a history of recurrent moderate to severe depression with multiple failed attempts at antidepressants discontinuation, continuation of antidepressant treatment during attempts at conception and pregnancy is reasonable. A tricylic, that is least likely to cause orthostatic hypotension (eg., nortriptyline) or SSRIs can be used. Use of shortacting SSRIs (paroxetine or sertraline) may be preferred over fluoxetine which has a longer half life. MAOI use should be avoided. Information regarding potential risk associated with the use of venlafaxine and bupropion is lacking. If a patient has history of severe recurrent major depression or prior postpartum depression and who is not on antidepressants, it is advisable to reintroduce antidepressants in the third trimester or during the early puerperium (Altshuler et al., 1996).

Some factors to be considered for each drug, when used in the pregnancy or during lactation include

- Teratogenesis
- Influence on foetal Growth
- Precipitation of premature labour
- Intoxication of the newborn
- Neonatal withdrawal syndrome
- Lactation effects
- Behavioral toxicity

Principles of drug management during lactation include using the lowest possible dose, avoiding depot preparations, avoiding multiple drugs, and knowledge about the pharmacokinetics of the drug, so that dosing can be decided in relation to breast feeding. Benzodiazepines should be avoided in neonates and premature infants and one must avoid long acting drugs.

Conclusion

Perinatal psychiatry, is an important field which merits the best service we can provide, in which the life and well-being of two individuals are at risk, one of them starting off on life's journey, the other

experiencing huge changes and life events. To manage perinatal psychiatry well, is to improve the psychiatric prognosis of the mother and reduce the risks for the future ill health of the child. To ignore it, is to jeopardise both.

Case of Premenstrual Syndrome

Ms. Gauri, is a 22 year old postgraduate student, with history of frequent mood swings. She has days in which, she is extremely irritable, angry, cries for no reason and is very sensitive. In her own words, on these days her friends avoid her. On further questioning, it appears that these symptoms appear about a week before her menstrual period and continue till her period is over. She also reports jitteriness, feelings of tension and feeling heavy and bloated. She often misses assignments and classes on these days, as she feels she cannot cope. She denies experiencing any of these symptoms on other days. She has no other medical or gynecological problems though she reports a low backache during this period.

Ms. Gauri appears to have a premenstrual disorder with predominantly depressive symptoms severe enough to interfere with her social and occupational functioning.

At this stage, the approach would be to do a prospective rating of her symptoms across two menstrual cycles to establish periodicity and the exact relationship to the menstrual cycle. She will also need a thyroid function assessment and a gynecological evaluation to rule out conditions such as fibroids or endometriosis.

Premenstrual syndrome

The influence of menstrual cycle on behaviour and mood has been described widely. In the 1960's, the first clinical descriptions of the Premenstrual Syndrome (PMS), were published by Katherine Dalton. Since then, there has been an upsurge in research on the PMS. Researchers have examined PMS from various perspectives, including biological, sociological, anthropological and as well. legal Premenstrual, behavioral and mood changes can be divided into three broad categories based on the magnitude of the changes, their occurrence in relation to the menstrual cycle and the degree of disability they cause.

A large majority of women (nearly 80-90%), experience some changes during the premenstrual period. Usually these changes are, somatic in nature and are mild in severity. In most women they do not cause any functional impairment and only help in heralding the onset of the menstrual period.

The PMS generally refers to a constellation of affective, physical, behavioral and cognitive changes that occur with a repetitive cyclical relationship to the luteal phase of the menstrual cycle and are present in most cycles. The symptoms may be of sufficient severity to cause distress and/or functional impairment. This syndrome equivalent of premenstrual changes, is found in nearly 4-5% of women and usually warrants treatment (Chandra & Chaturvedi, 1989).

Premenstrual exacerbation of a pre-existing disorder

A number of medical conditions such as asthma, allergies, irritable bowel, migraine and epilepsy might be exacerbated during the premenstrual period. Some psychiatric disorders, especially

depression and occasionally psychosis may exacerbate just prior to the onset of menstruation. Occasionally, impulsivity, aggressiveness, irritability or hostility occurring as symptoms of a psychiatric disorder might get exacerbated rather than the overall condition.

The premenstrual syndrome (PMS) has often been described as a heterogeneous disorder. The heterogeneity, mainly relates to the diverse ways in which it presents, the combination of various experiences and the differences found in various population groups. Though the changes described in the premenstrual syndrome usually occur in the premenstrual phase, the duration is variable and may range from 1-7 days prior to the periods with remission within one or two days of onset of the menstruation. n some women, these changes can also surface during the ovulation, and may persist during both the early and late parts of the luteal phase. By virtue of their disabling effects, premenstrual changes and more often the syndrome can interfere with women's performance at work and influence adversely their interpersonal relationships. For some women (nearly 5%) these can be very disruptive and will necessitate treatment (Chaturvedi et al., 1993).

Clinical features

Following clinical features appear in most menstrual cycle. These symptoms usually start a week before the onset of menstruation and subside within a day or two after the onset (Chaturvedi et al., 1993; Chaturvedi et al., 1995).

- Markedly depressed mood, feelings of hopelessness, or self-deprecating thoughts.
- Marked anxiety, tension, feelings of being 'keyed up', or 'on edge'.
- Marked affective lability (e.g., feeling suddenly sad or tearful or increased sensitivity to rejection).
- Persistent and marked anger or irritability or increased interpersonal conflicts.
- Decreased interest in usual activities (e.g. work, school, friends, hobbies).
- Subjective sense of difficulty in concentrating.
- Lethargy, easy fatigability, or marked lack of energy.
- Marked change in appetite, overeating, or specific food cravings.
- Hypersomnia or insomnia.
- A subjective sense of being overwhelmed or out of control.
- Other physical symptoms, such as breast tenderness or swelling, headaches, joint or muscle pain, a sensation of 'bloating', weight gains.
- The disturbance markedly interferes with work or school or with usual social activities and relationships with others (e.g., avoidance of social activities, decreased productivity and efficiency at work or school).

Symptoms and Subsyndromes in the PMS

Depression (85%), irritability (80%), mood swings and anxiety (60%), have been reported to be the commonest symptoms in women with PMS. These figures indicate the high prevalence of mood disturbances. The second most commonly occurring group of symptoms is somatic in nature and their prevalence varies from 40-60%. Cognitive symptoms have been described in 40%, and symptoms indicating water retention in 60%.

Symptoms of Premenstrual Syndrome may be grouped as follows:

Mood Changes

- Irritability, 'irrational anger outbursts'
- 'At war with the world'.
- Mood swings.
- Feelings of guilt.
- Suicidal ideation.
- Depression, inability to cope.
- Anxiety being on edge

Somatic Symptoms

- Decreased energy levels.
- Fatigue.
- Tiredness and lethargy.
- Decreased concentration and slowed thinking.
- Pain.
- Headache.
- Sleep disturbances.
- Insomnia or hypersomnia.
- Changes in appetite.
- Craving for certain foods specially carbohydrates and spicy food.
- Changes in sexual desire

Change in Behavior

- Lack of self control.
- Proneness to easy crying.
- Hostility and occasional violence rejection sensitivity.
- Lack of interest in socialization

Behavioral symptoms seem to cause the maximum disability and because of these and other distressful symptoms some women might also abstain from work.

Cognitive Changes

- Muddled thinking.
- Impairment in memory.
- Poor judgment, indecisiveness and impairment in concentration.
- Being more accident prone and clumsy

Changes Associated With Water Retention

- Engorgement of the breast with pain
- Weight gain
- Clothes and shoes becoming tight
- Swelling of the feet
- Glaucoma with pain in the eyes and blurred vision
- Pain in the legs and abdominal pain
- Abdominal bloating

Course and Prognosis

Several studies, have shown an increased prevalence of affective disorders in women with premenstrual affective changes. Women with premenstrual changes, seem to have a higher lifetime prevalence of affective disorders. The incidence of postpartum mood changes is also higher in this group. Premenstrual changes worsen with age and are reported to be maximum in women in their third decade and gradually subside with menopause. However, women with PMS have been found to have more mood related problems in the perimenopausal period. Apart from affective disorders, women with PMS have an increased prevalence of alcohol abuse and eating disorders.

Management

The initial step in treatment of PMS remains the confirmation of definite PMS by a prospective rating at least two cycles. This is important for two reasons:

- Firstly, it differentiates between premenstrual exacerbations of pre-existing disorders and a premenstrual syndrome.
- Secondly, it helps the clinician in identifying which sub syndrome or syndromes characterize the PMS in the identified patients.

It has also been reported that, only in 20% of women (who retrospectively report a PMS) the diagnosis is confirmed by prospective ratings. Once a diagnosis of PMS is reached, several gynecological and medical conditions that might mimic a PMS need to be ruled out.

The patient requires a thyroid function assessment and if possible a hormonal analysis that includes estrogen, progesterone and prolactin levels at the luteal phase. Hypoglycemia due to any other cause might present with similar symptoms. Fasting as well as post prandial glucose levels may be necessary. In some cases a glucose tolerance test may be required. Gynecological conditions such as endometriosis, polycystic ovarian disease and uterine fibroids should be ruled out. A careful psychiatric evaluation to detect the presence of a comorbid affective disorder such as major depression, bipolar disorder and dysthymia is also necessary (Ling, 2000).

Pharmacotherapy

One common observation about PMS treatment is the high rate of placebo response and the inconsistency of treatment results. Despite the methodological flaws, there are some findings, which are consistent. The following pharmacological agents have been tried for the treatment of PMS:

- Progesterone
- Oral contraceptives
- Gonadotrophin releasing hormone
- Synthetic androgens like danazol
- Prolactin inhibitors like bromocriptine
- Diuretics
- Atenolol
- Prostaglandin antagonists like mefenemic acid
- Prostaglandin precursors like gamma linoleic acid, evening primrose oil
- Pyridoxine (Vitamin B6)
- Vitamin A
- Magnesium

Antidepressasants and anxiolytics

In view of the frequent occurrence of depression and anxiety in PMS, a number of antidepressants and antianxiety agents have been tried with varying degrees of therapeutic success. Studies have shown efficacy with nortriptyline, clomipramine and fluoxetine and Sertraline. Classical tricyclic antidepressants have also been used in some studies with good results. Among the anxiolytics, alprazolam is the drug which has been most commonly used and has shown a global improvement in all aspects of PMS. Buspirone, used in the treatment of PMS shows improvement in irritability, fatigue, social functioning and pain.

Over 30 reported studies and several ongoing studies, have documented the efficacy and tolerability of serotonin-enhancing drugs in up to 70% of women with severe PMS.

In most of the drug trials with SSRIs, the does needed to achieve response was relatively low. In one of the dose-response study in which fluoxetine was used, it was documented that there was no advantage in increasing the dose beyond 20 mg/day and that patients taking 60 mg/day had a significantly higher incidence of side effects.

The onset of response with SSRIs in PMS is very rapid, sometimes as short as 1 to 2 days, which has prompted several investigators to consider intermittent (luteal phase only) rather than continuous dosing of the medication. To date, the effectiveness and tolerability of the intermittent dosing regimen have been confirmed for sertaline, fluoxetine, and citalopram, with several additional randomized placebo-controlled trials currently under way. Of note is the recent trial with citalopram which showed that not only was the drug more effective than placebo, but intermittent dosing was more effective than continuous dosing (Erikson et al., 2002).

MANAGEMENT OF PMS

Rule out a psychiatric illness or an endocrinological problem

↓

If hormonal changes in late luteal phase use progesterone or oral contraceptives

↓

Daily charting of symptoms prospectively over two cycles

↓

Mild premenstrual changes

↓

Reassurance
Exercise
Dietary changes

↓

Use nonpharmacological measures

↓

No change
(Proceed as for PMS)

↓

water retention	anxiety	depression	non specific	pain	mood swings
Diuretics Atenelol	Buspirone Alprazolam	SSRIs TCAs	GLA Vit.E, B6, Mg	Mefenemic acid	Antidepressants lithium

↓

If no change
Try GnRH or synthetic androgens

Severe changes (PMS)

↓

Establish predominant symptoms or symptom groups

↓

Conclusions

Premenstrual distress occurs in many women, but some have a distressing premenstrual syndrome. Though the exact etiology is still not well understood, with appropriate treatment substantial relief in PMS symptoms can be obtained. Life style alterations also form an important step in the management of PMS.

Anxiety and Depressive Disorders in Elderly population

S. Bharath

B. Somashekar

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Depressive disorders in elderly Population

Introduction

The process of aging becomes evident, from the fourth decade onwards. As individuals age, they are more likely to suffer from disease, disability, and the side effects of drugs, all of which, when combined with the decrease in physiologic reserve, make the older person more vulnerable to psychological, environmental, pathologic, and pharmacological challenges. In addition the disease presentation in the elderly, compared to the middle-aged patients, is often atypical.

Depression and anxiety, which are commonly observed in the elderly patients, are potentially treatable conditions, irrespective of the cause. Early recognition, assessment and treatment of depression and anxiety, lead to significantly improved quality of life, of these elderly patients.

Case of depression in an elderly diabetic and hypertensive patient

Ms.Shanti, 65 year old, married lady, is a known diabetic and hypertensive, since the past 15 years and is on concomitant antidiabetic and antihypertensive treatment. She lives with her son and daughter-in-law who are very supportive. Episodically over the past 3 years, Shanti has been observed to be very restless, often sighs, does not show any interest in the household activities, sleeps poorly and refuses to eat. In addition, she often becomes irritable, complains that her son is not taking care of her adequately and demands repeated medical consultations. During these episodes, each of which last for 3-5 months, she is unable to recall the names of her son and grandchildren correctly. She is also unable to locate specific places in the house. In between the episodes, when she is relatively symptom free, though her memory appears to be reasonably adequate, overall it is poor when compared to the memory of her older sister, who lives nearby and is healthy. During the initial episodes, she consulted a physician, who attributed her behavior to hypo or hyperglycemia. He did not consider depression, as Shanti never complained of being depressed, wanting to die, being worthless or, waking early in the morning during these episodes. Later the physician referred her to a psychiatrist. Shanti was diagnosed to be suffering from 'Recurrent Depressive Disorder' and was initiated with sertaline (50 mg/day) treatment. Though, sertraline treatment led to some improvement, Shanti continues to suffer from repeated depressive episodes, each lasting for 3-4 months. She is symptom free for only a brief period of about 3 months in between these episodes. This has made her even more dependent on her son than what she might have been otherwise.

Case of depression in a bank manager

Mr. Chauhan, is a 55 year old man, who has led a very active professional life as a senior manager of a nationalized bank. For him profession has been the pivot of his life and he enjoyed his role very much. Two years back, Chauhan, opted for voluntary retirement as he planned to avail the cumulative monetary benefit and take up another similar job in a private bank. After retirement, he realizes that re-employment at the age of 56 year especially in private banks, is not feasible. Despite being financially comfortable and having the support of his working wife and two studying sons, Chauhan feels listless and amotivated to do any job around the house. He finds it difficult to go out and meet friends and to look for job opportunities in other areas related to finance. He is unable to socialize and

participate in religious activities. He had always complained of not having time to go to places and visit people when he was working. Now he does not want to travel or visit people though he has plenty of time at his disposal. He has very poor appetite, wakes up early in the morning at 2 am and has to be coaxed to shave and wear fresh clothes. His wife observes him to be least interested, even in viewing television. She feels, that he is developing some major illness due to old age, which is responsible for his symptoms like, excessive tiredness, inability to go out in search of job, inability to socialize etc. She also recognizes the fact that he has no hobbies and that his life had centered around his 'job' earlier. At the same time she is worried about him having any physical illness like cancer, which is making him so tired and run down.

These two typical elderly patients are suffering from Depression.

Depression and anxiety in elderly

Globally, the population is growing older, as there has been a gain of 30 years of life expectancy in this century (Lebowitz et al., 1998). The awareness of the disabling consequences of depression in these elderly patients, has been increased by the Landmark report of World Health Organization on the 'global burden of disease' (Murray et al., 1996). With the increased usage of tools of contemporary neurosciences, there has been a significantly enhanced understanding of the pathophysiological and etiological mechanisms of depression (Duman et al., 1997; Melzter et al., 1998). These three factors have contributed significantly to realization of depression and anxiety in elderly patients as a leading public health issue.

Epidemiology

Despite the controversy over the prevalence of depressive disorders in elderly population in the community, whether it is lower than in the younger population or not (Reiger et al., 1984), community surveys studies have shown that 10%-25% of the elderly general population has depressive or dysphoric symptoms. 1%-5% of the elderly in the community suffer from depressive disorders, more common being dysthymia (2%-8%). Most studies recognize that the prevalence varies with the instruments used to assess depression, gender, place of study and the comorbid physical conditions. A higher prevalence has been reported in females, urban areas, in long-term care facilities (as high as 42%) and those with medical illnesses (12-23%) (NIH Consensus Conference, 1992).

Community surveys in India have concluded that depression is the most common psychiatric disorder (24%) in late life (Nandi et al., 1975; Ramachandran, 1980; Venkoba Rao & Madhavan, 1982).

Depression in the elderly is often overlooked due to various factors.

- Normal aging results in universal slowing of all mental activities. The symptoms of depression such as psychomotor retardation and decreased interest may wrongly be attributed to aging or physical conditions common in the aged, if the interviewer is not in pace with elderly and inquire other symptoms of depression both with the patient and the family members.

- Concomitant medical and neurological illness may result in wrong assessment - e.g. motor

symptoms of depression may be mistaken for symptoms of Parkinson's disease such as bradykinesia and expressional-less face.

- The disabilities such as hearing and visual impairment may hinder proper assessment.

- Transferencial issues may lead to the elderly under reporting personal experiences and suffering especially sadness, as interviewer will be invariably younger.

- Counter-transferencial issues may result in poor assessment of depression, e.g. the interviewer may feel uncomfortable to ask sensitive questions related to suicidal behavior, substance use and sexual functioning.

- It is known that elderly depressives often report of bodily complaints than depressed mood.

Etiology

Contributory factors

Certain factors make the elderly a unique population prone to depression and anxiety.

1. Commonality of physical illnesses in late life can predispose the elderly to depression, e.g. cerebrovascular diseases, hypertension, myocardial infarction, diabetes, arthritis, malignancies, sensory impairment, etc.

2. Drugs used for the medical conditions, e.g. some of the earlier antihypertensive were known to produce depression.

3. Life events which increase the vulnerability to depression and depressive reactions, e.g. retirement, offsprings leaving home, death of spouse and friends, restrictions in mobility and disability, social isolation etc.

Psychosocial and biological factors: These factors are more implicated in the various types of depression in late life than genetic factors.

Neurodegenerative and vascular factors: A small group of depressives with an onset in late life is shown by imaging studies (MRI) to have a neurodegenerative basis with multiple infarcts and significant cognitive disturbances. The term 'vascular depression' is used for this type of depression in late life.

Clinical features

Depression as a clinical syndrome is similar in the old and the young. Studies spanning over five decades have failed to identify clinical features specific to depression of late life (Brodaty et al., 2001). Elderly depressives report affective, cognitive, behavioral, motor and biological symptoms like other adult depressives.

However, certain trends are discerned in depression of late life. Motor symptoms in the form of retardation or agitation are common. Agitation often manifests in the form of pacing, being restless, distractible, fidgety, being uncooperative for interviews and appearing confused (Venkoba Rao & Madhavan, 1982). A retarded elderly is apathetic, complains of lack of energy, every activity being an effort, not completing even simple personal tasks and not wanting to be involved in house activities (Gurland, 1976).

The elderly may not readily report sadness but instead may report anxiety, irritability, and fear of having physical illness. Crying spells are rare so also reporting of guilt or suicidal ideas. Bodily symptoms and persistent pain are common and various studies have recognized this. Among the biological functions, greater weight loss is seen among the elderly depressives.

Cognitive symptoms manifest in the form of poor concentration, muddled thinking, indecisiveness, loss of memory and may present a picture of dementia or confusion.

Psychotic symptoms are more common in severe depression in the elderly and may present as delusions of ill health, persecution and rarely guilt (Venkoba Rao & Madhavan, 1982). Milder forms of depression and like dysthymia present with irritability, complaining against family members, feeling neglected, pessimistic, demanding and clinging behavior. These are often perceived by others as signs of 'becoming old' and so the due attention and treatment are not provided.

Comorbid conditions

Depression is a common comorbid condition in some of the old age illnesses influencing the course and outcome of the condition. They are,

1. Depression associated with dementia especially Alzheimer's dementia: As much as 23% of persons with dementia have depression. Sometimes depression is the forerunner of dementia (Alexopoulos, 1991).

2. Depression associated with Parkinson's disease: Depression could be secondary to or associated with Parkinson's disease. The presence of bradykinesia and the mask like face often make the recognition of depression and its treatment difficult.

3. Depression associated with stroke: Both Depression and Dysthymia are common in stroke (20-40%). Lesions of the left cortical and subcortical region and those with nonfluent aphasia are more associated with depression.

4. Depression associated with cardiovascular illness: A complex relation exists between physical illness (especially cardiovascular illness) and depression. Myocardial infarction and certain serious medical disorders increase the risk for depression and major depression increases the risk of re-infarction and death in people who have had recent myocardial infarction. The interaction between depression and cardiovascular disease may reflect the pathophysiological impact of mood disorder mediated by elevation in cortisol, peripheral catecholamines and altered platelet function besides poor adherence to treatment.

It has been already mentioned above that research indicates that cerebrovascular disease may result in depression in late life and late onset depression may be a forerunner of Alzheimer's disease and Parkinson's disease. Thus effective treatment may prevent or delay the onset of Alzheimer's disease and Parkinson's disease.

Course and Prognosis

Studies on natural history of affective disorder show that the episodes of depression occur more frequently and last longer with age. The peak incidence of new cases of depression is in young age and declines with age.

Depression in late life is often a challenge. Total remission may not be possible in a significant number of elderly depressives in comparison to the young people. Reduction in the severity of depression and normalization of activities to the extent possible are feasible goals.

Management

'There is a need to add Life to Days rather than Days to Life'

Quality of life of an elderly improves with treatment of depression. This is the most important reason why depression in late life needs to be recognized and treated. There are other snowballing effects in treating a depressed elderly person.

General Principles of Management

- Confirmation of diagnosis.
- Identification of type of depression: retarded or agitated.
- Assessment of physical and psycho social status of the patient.
- Choice of medications to be used.

Assessment

It is essential to bear in mind that depression in older patients is often associated with physical illness. Factors such as cognitive functions, concomitant physical illness, psychological functioning and social support should be taken into consideration when assessing depression in elderly.

Assessment should include

- History including family history from the elderly and a close relative.
- Assessment of Psychosocial status including support, economic independence, decision making role in the family etc.
- Recent Life Events.
- Medical History and Physical Examination.
- History of Medication for Physical conditions.
- Mental Status Examination (serial when necessary).
- Comprehensive Laboratory Investigations

1. Cardiac, Vascular, Renal, Hepatic parameters.
2. Nutritional Status.
3. Endocrinological investigations mainly thyroid status.

- Cognitive assessment and imaging studies such as MRI, if there are vascular risk factors..

Most elderly patients can be treated on an out patient basis with regular follow-ups especially in the initial phase of the treatment. Hospitalization is to be avoided whenever possible as it may be difficult for the elderly to adjust to a new hospital environment and ensuing cognitive difficulties may further complicate the recovery from depression. It is indicated in patients with suicidal behavior, when medical or pharmacological factors interfere in treatment of depression and in those who fail to recover with adequate trial of antidepressant treatment.

Pharmacotherapy

In principle, if the depression is a complication of another disease (e.g. hypothyroidism) or use of medications (e.g. antihypertensives), treatment of the primary condition is indicated. If depression coexists with another physical condition (e.g. Parkinson's Disease) treatment of both simultaneously are necessary. The depressive symptomatology and the type of physical condition should guide the selection of appropriate antidepressant.

The pharmocokinetics of a psychotropic drugs are different in elderly due to various physical changes such as relative increase in body fat, decrease in total plasma volume, and decrease in total body water and extra-cellar fluid. As a result, the volume of distribution of fat-soluble drugs (e.g. tricyclics) is increased and excretion is delayed. Larger portion of drugs with high protein binding remain unbound and pharmacologically active due to low plasma albumin levels. Due to decrease in the motility and absorbing cells, both absorption and excretion are delayed.

For the above-mentioned reasons, initiating half of the adult dose is recommended in elderly. However a standard prescription pattern is difficult. Hence, drug prescription should be individualized based on the physical condition and previously tolerated drugs and their doses in elderly. Elderly may have difficulties in swallowing, especially if they have concomitant physical disease (e.g. Parkinson's disease). In such situation using liquid preparation can be considered.

To avoid sudden and drastic increase in blood levels of drugs in elderly, divided doses may be used even though single dose is recommended in adults. Sudden and transient increase in blood levels with single dosing may cause confusion, hypotension, fall, or other side effects. However the prescription should be as simple as possible to avoid wrong use of medication and minimize side effects.

Although, monotherapy is ideal, it may not be possible to adhere to the rule. Use of adjunctive medications such as anxiolytics and antipsychotics has several advantages whenever appropriate. For patients with agitated depression, Selective Serotonin Reuptake Inhibitors (SSRIs) group of antidrepessants like sertraline 25-50 mg/day may be initiated and slowly increased to the tolerated dosage. Benzodiazipines such as clonazepam 0.25-0.75 mg/day may be used as adjuncts for control of agitation, anxiety and sleep normalization. Once these are achieved the benzodiazepine is tapered and stopped.

Combination of two types of antidepressants a tricyclic and SSRI though done for augmentation in resistant depression, should be best avoided in the elderly due to higher risk of side effects.

Choice of antidepressants

Pharmacotherapy is the mainstay in the treatment of elderly.

Various antidepressants used in elderly with depression and anxiety are

1. Tricyclics anti depressants (imipramine, amitriptyline, nortriptyline, doxepin and antidepressant)
2. Selective Serotonin Reuptake Inhibitors (SSRIs) (fluoxetine, sertraline)
3. Monoamine oxidase Inhibitors (MAOIs) (moclobemide)
4. Newer miscellaneous antidepressants (venlafaxine, mirtazapine)

There is no ideal antidepressant drug for elderly patients with major depression. All classes of antidepressants are equally effective in elderly but in general are poorly tolerated as compare to younger patients. The choice of antidepressant depends on the symptoms, action of drugs, side effects, previous history of response and cost.

The most commonly used antidepressants in India, are tricyclics and SSRIs. Tricyclic antidepressants have anticholinergic and cardiac side effects. They also produce hypotension. Among the tricyclics, amitriptyline and imipramine should be avoided whenever possible in elderly patients (especially when depression is diagnosed for the first time in elderly) because of potent anticholinergic and cardiovascular side effects including hypotension. Dothiepin, doxepin and nortriptyline are preferred.

SSRIs are gaining increased acceptance in the elderly patients because of their better side effect profile and are used as the first line antidepressant in late life depression. However, they are not totally free of side effects. SSRIs can cause agitation, anxiety and sleep disturbances.

One should become familiar with one or two agents for the treatment of patients with predominant psychomotor retardation (e.g. fluoxetine, sertraline) and for those with predominant agitation (e.g. nortriptyline, trazodone). Careful follow-up is required to anticipate and minimize anticholinergic side effects, orthostatic hypotension, sedating effects, confusion, cardiovascular complications, and drug overdose with suicidal intent. Adverse drug reactions should not be assumed to be due to the aging process.

Although not very popular in India, when used continuously the reversible monoamine oxidase inhibitors (RIMA) like moclobemide, may be useful when other antidepressants are ineffective. Monoamine oxidase inhibitors should not be used in combination with the tricyclic compounds.

Newer antidepressants like venlafaxine and bupropion have also been tried in the elderly patients. Adequate number of double blind trials of various types of antidepressants in varying doses in the depressed elderly is lacking and hence often the decision on the type and dose of antidepressant is based on clinical experience and personal preference than on evidence based research.

Electroconvulsive Therapy

Electroconvulsive therapy (ECT) is effective and usually well tolerated by elderly patients who remain severely depressed despite drug treatment, particularly if they also have psychotic features and psychomotor retardation (Sackeim, 1998). Recent evidence suggests that ECT may be more effective in elderly than younger patients. Presence of cognitive dysfunction itself is not a contraindication for the use of ECT. Cognitive side effects may be less with unilateral ECT.

Psychotherapy

Research evidence is slowly emerging on non-physical types of treatment of depression in the elderly. Psychoeducation, Cognitive Behavioral Therapy (CBT) and Insight Oriented Therapy have been found to be effective (Teri and McCurry, 2000).

In elderly depressed patient with comorbid medical conditions treatment modalities life coping strategies, problem-solving skills and relaxation have been found to be effective.

Conclusions

Ignoring and assuming 'depression' as a part of aging results in cumulative loss of revenue and expenditure of avoidable health resources. Effective use of pharmacotherapy & psychotherapy leads to improvement in the quality of life of these depressed elderly patients.

Anxiety disorders in elderly population

Asthma or Panic - a case report

Mrs Rajlaxmi, a 65 year old lady presented with history of brief episodes of shortness of breath, palpitation, sweating and tremors. During the attack she felt as if her heart was sinking and life would come to an end. She was a known case of asthma stabilized on bronchodilators. On investigation her chest X- ray and electrocardiogram did not reveal any abnormality. On further evaluation her thyroid function were abnormal and a nodule was detected on clinical examination. Her respiratory function tests were within normal limits during the attack. No other endocrinal, cardiovascular, central nervous system or respiratory disorder could be detected on clinical examination, radiological evaluation or biochemical investigations.

Clinical history in this case was suggestive of either bronchial asthma or panic disorder. Since her respiratory function tests were normal during the attack, she was diagnosed to have panic disorder. In view of late onset of panic disorder she was further investigated and a carcinomatous nodule was detected in her thyroid gland along with thyroid function abnormality. Apart from this she was also taking xanthine group of drugs for bronchial asthma that could have aggravated her anxiety symptoms.

Anxiety disorders in elderly

Anxiety disorders in elderly individuals are commonly associated with various medical disorders that make clinical diagnosis difficult by confounding clinical symptoms. Often anxiety disorders occur in early adulthood, tend to be chronic, interspersed with remissions and relapses and usually continue into old age (Sheikh and Salzman, 1995).

Epidemiology

Late onset anxiety disorder seems to be rare. There is scant data on prevalence and incidence of anxiety disorders in elderly but overall prevalence seems to be lower than that in younger population (Blazer et al., 1991).

Generalized anxiety disorder and phobia are most common among the elderly while panic disorder appears to be rare. It persists either from younger age or arise in the context of another psychiatric or medical disorder (Flint, 1994)

In females agoraphobia and obsessive compulsive disorder (OCD) may occur as primary disorder in old age for first time, whereas in males simple phobia and OCD are more common (Flint, 1994).

Etiology

Exact etiology of anxiety disorder in elderly is unclear. Existing literature hints towards some medical, physical and psychosocial problems that are common in this age group.

Among medical disorder hyper and hypothyroidism, congestive heart failure, pulmonary embolism, angina, arrythmias, chronic obstructive pulmonary diseases, pneumonia, neoplasm, Parkinson's disease and substances such as alcohol, steroids, thyroid preparations, anticholinergics and antidepressants have been implicated in anxiety symptoms in elderly.

Elderly population is also predisposed to significant psychosocial problems. Retirement may lead to social isolation, loss of direction and personal identity. Death of spouse may lead to prolonged bereavement and fear of separation and abandonment may lead to anxiety symptoms. Anxiety in elderly is mainly due to object loss and fear of loss of external support rather than intrapsychic conflict.

Real life burdens such as financial problems, crime and fear of personal safety, caring for ill spouse, poor housing and difficulty in obtaining medical care aggravate anxiety in these individuals (Sheikh & Salzman, 1995).

Clinical features

Clinical presentation of anxiety disorders may be different in geriatric population than that in younger population. Research data suggests that sub-syndromal states and somatic anxiety are more common than full blown clinical picture and cognitive symptoms respectively in this group. (Sheikh and Salzman, 1995).

Agitation

Agitation is a common presentation of anxiety in old age. However in geriatric population it may be manifestation of underlying depression or acute medical illness especially in dementia patient. A thorough medical history and clinical examination may reveal the true diagnosis.

Panic disorder in elderly population is usually continuation of younger age onset. Though, late onset panic disorder has also been reported. Existing data suggest that panic disorder in elderly population may present with fewer symptoms, less avoidance and lower scores on somatisation measures (Luchins & Rose, 1989).

Late onset agoraphobia is commonly due to physical illness (Lindsay, 1991). Presence of dentures and tremors may lead to eating or writing phobia in public. Crime and various medical dental procedures are also reported to cause phobias in this population.

A recent report, documents late onset of Posttraumatic stress disorder (PTSD), among survivors of 1988 American earthquake (Goenjiane et al., 1994). They report less re-experiencing and more hyperarousal symptoms in older patients than younger patient.

A high prevalence of depression and physical illness has been reported in elderly with generalized anxiety disorder (GAD) (Lindsay et al., 1989).

Studies comparing OCD in young and elderly population are scant.

Comorbidity

Anxiety symptoms are often masked, under-diagnosed, misdiagnosed or exacerbate due to various physical and psychiatric comorbidity. Among psychiatric disorder depression occurs in almost one third to half of the patients of anxiety disorders. Alcohol dependence syndrome is highly prevalent with panic and phobic disorders. Cognitive decline may occur in some of the patients. Sensory impairment and central nervous systems disorder (e.g. delirium and stroke), cardiovascular disorders (e.g. angina and arrhythmia), respiratory system disorder, (e.g. bronchial asthma and pulmonary embolisms), endocrine disorders (e.g. hypo or hyperparathyroidism or diabetes) are frequent comorbid conditions (Lesser, 2000).

Management

Principles of management of anxiety disorders in elderly are largely derived from extensive research data available on young population. Data on elderly population is inadequate to draw any conclusion.

The first step in management of these patient is evaluation of medical illness by history, clinical examination and relevant investigation, since physical illness are highly prevalent in this group and may manifest as anxiety disorder. If patient is receiving other medications, assessment of adverse effects and interactions of these medications becomes necessary.

Clinical assessment of cognitive function should be done routinely in all the elderly patients since anxiety symptoms may be manifestation of early onset dementia.

Finally, psychosocial problem and comorbidity should be assessed to provide holistic treatment.

Both pharmacological and non-pharmacological treatment may be considered singly or in combination depending upon nature of problem.

Pharmacotherapy

Age related physiological changes in elderly affect absorption, distribution, protein binding, metabolism and excretion of drugs. Elderly patients may have higher plasma levels of drugs than younger patients due to these physiological changes. Therefore they need half to quarter dose than needed for younger adults. Once these drugs are tolerated well, doses may be increased to get optimum therapeutic response.

Benzodiazepines

Benzodiazepines may be used in management of generalized anxiety disorder (GAD). Drugs with a short half-life such as alprazolam, oxazepam, lorazepam and those metabolized by direct conjugation in the liver are preferred as they are less likely to accumulate. Very short acting benzodiazepines can cause confusion, agitation and anterograde amnesia. Long term use of benzodiazepines may lead to dependence. Some very old patients may develop disinhibition, and experience agitation or aggression. Adverse effects of benzodiazepines include sedation, cognitive dysfunction, impaired psychomotor performance, and cerebellar dysfunction. There is increased risk of fall with short half-life medication. Rebound withdrawal may occur but it is less severe than in younger patients.

In panic disorder, high potency benzodiazepines like alprazolam and clonazepam appear to be efficacious for symptom control (Ballenger et al 1988). They have an advantage of rapid onset of effect compared to antidepressants, which typically take two to four weeks for therapeutic effects. However, benzodiazepines should not be used for more than few weeks in elderly because of several undesirable side effects and abuse potential. Benzodiazepines may be combined with SSRIs (Selective Serotonin Reuptake Inhibitors), in patients who might be extremely anxious. After few weeks, benzodiazepines may be tapered, while continuing the SSRI for at least 1.5 to 2 years, at which point tapering may be considered (Sheikh and Salzman, 1995).

Antidepressants

1. Panic disorder: Tricylic antidepressants (TCAs) like imipramine and monoamine oxidase inhibitor (MAOI) like phenelzine have been documented to be effective in panic disorder and agoraphobia (Mavissakalian and Michelson, 1986). More recently, several researchers have demonstrated efficacy of SSRI in panic disorder such as fluoxetine, sertaline and paroxetine (Ohrstrom et al., 1992). Due to the undesirable side effects of imipramin and amitriptyline and the MAOIs in the elderly, drugs like dothiepin or doxepin or nortriptyline or trazodone and SSRIs are preferred (Ballenger, 1994). SSRIs are often the first line of treatment, as they seem to be much better tolerated by the elderly compared to imipramine and phenelzine. Half of the initiating dose for younger patients may be suitable to begin with in this population, and gradually increase to a therapeutic dose as tolerance of the patient improves. MAOI may be used in refractory case of panic disorder.

2. Social phobia: There is evidence that both irreversible and reversible MAOI are effective in social phobia (Liebowitz et al., 1992). Most recently an open label study suggested the effectiveness of fluoxetine in social phobia (Van Vliet et al., 1992).

3. Posttraumatic stress disorder (PTSD): Tricyclic antidepressants and SSRIs have shown mixed results in PTSD. (Davidson et al., 1990) Cognitive behavioural therapy for PTSD, consisting of imaginal and in vivo flooding, thought stopping or guided self dialogue has been found to be benificial in short-term management (Steketee & Foa, 1987). The management of PTSD is best provided in the context of a supportive therapeutic relationship with pharmacotherapy for symptomatic control.

4. Obsessive compulsive disorder: Several placebo controlled studies, have demonstrated efficacy of tricyclic antidepressant, clomipramine (Insel et al., 1983), and SSRIs such as fluoxetine, sertaline and fluvoxamine (Greist et al., 1992) in young population. But because of undesirable side effect profile of clomipramine, SSRIs are preferable in the elderly. A trial of at least 10 to 12 weeks of a particular SSRI is indicated before one can deem it to be a failure and consider alternative strategies. For those patients who do not respond to such a trial, switching to another SSRI or the addition of buspirone or clonazepam may be considered. Behaviour therapy may be combined.

Antihistamines

Mild anxiety symptoms, may be managed by antihistamines, like hydroxyzine and diphenhydramine for short periods (Sheikh & Salzman, 1995).

Beta-blockers

Beta-blockers may be useful in managing somatic anxiety by blocking autonomic reaction, commonly associated with anxiety (Tyrer and Lader, 1974).

Anxiety and agitation associated with dementia

Benzodiazepines, neuroleptic agents, SSRIs, trazodone, buspirone, beta-blockers and anticonvulsant medications have been used for agitation in dementia.

Benzodiazapines may be used for short-term because of their sedating effect. However, long-term use may lead to several adverse effects like confusion, incoordination or paradoxical reaction. Short acting agents like lorazepam or oxazepam may be better due to less risk of accumulation.

A review of studies, on effectiveness of neuroleptic agents, in the treatment of agitation suggests moderate efficacy with considerable concern of side effects when they are used (Salzman, 1990).

Beta-blockers have been found to be promising in some case reports especially in the cases that are refractory to antipsychotics or benzodiazepines (Petric & Ban, 1981). However, these are generally contra indicated in several medical conditions that are prevalent in elderly population. Several case reports suggest efficacy of trazodone, SSRIs and carbamazepine in these patients (Sheikh & Salzman, 1995).

Psychosocial intervention

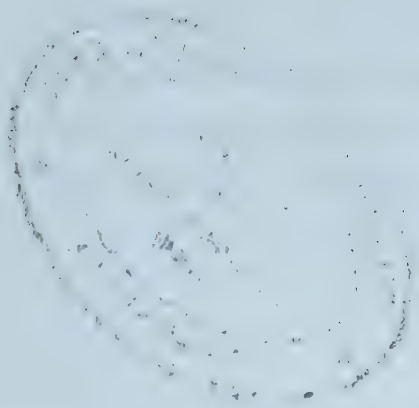
It may be appropriate for:

- Patient who have substantial psycho social problems such as bereavement, real life burden, social isolation, behavioral problems, ect.
- Patient who are unable to tolerate side effect of medication.
- Patient who are non-compliant for medication.
- Patient who show inadequate response to medication.
- Patients who prefer psychotherapy to medications.

Severe cognitive decline may be contraindication to cognitive behaviour therapy. Current strategies of psychotherapy of elderly patients are largely derived from effectiveness data in younger population. However, relaxation training, exposure, cognitive restructuring, education about illness and environmental manipulation have been reported to be effective in elderly population as well as by various studies. (Sheikh & Salzman, 1995).

Conclusion

Like for the very young, the aging body & mind are in many respects uncharted territory when considering psychopharmacotherapeutic interventions. The therapeutic decisions for the elderly should be made only after considering the presence of other medical disorders, other non-psychotropic agents, expectable changes with aging & a decreased functional reserve in certain organ systems (e.g., the brain or the kidney).



Anxiety and Depression associated with Stroke and Myocardial Infarction

Niraj Ahuja

Depression and anxiety after stroke

Case of Post Stroke Depression

Ms.Leena, a 58 year old, unmarried teacher, living with her brother, consulted a psychiatrist because she had difficulty in thinking, concentrating and remembering past events. In addition, she complained of declining interest in her teaching activities and inability to sleep. These problems originated 8 months ago, when Leena a known hypertensive was admitted to the hospital, with complaints of weakness in the right half of the body and difficulty in speech. She was treated for this episode of stroke in the hospital for a brief period and was subsequently managed at home. Despite the initial rapid improvement, mild weakness in the right lower limb persisted. Since the past 4 months she started complaining of being awake for 2 hours every night before she could sleep, awoke early and was not able to go to sleep again. She would frequently lie awake with her mind brooding over the fact that she could not walk properly due to the residual weakness and felt handicapped. Her brother informed, that she had stopped going to the school and also avoided family functions, claiming that she was not feeling well and she generally appeared sorrowful to him. She often found herself in tears on thinking about her condition. Over the last few months, she had become even more miserable and had lost all hopes for any improvement. She even felt joyless and fatigued. Her weakness in the right lower limb persisted as she refused to comply with physiotherapy.

Leena was diagnosed to be suffering from Post Stroke Depression.

Post Stroke Depression

Stroke is defined as the sudden loss of blood supply to an area of the brain resulting in permanent tissue damage. It is the most common neurological disorder, accounting for nearly half of all neurological admissions in the US. It is the third leading cause of morbidity and mortality in the US (after heart disease and cancer), with an estimated annual incidence of 300,000 to 400,000. It is more common in men and incidence rises steeply with increasing age. Among stroke patients, the ischemic subtype accounts for 80-85% of cases, while the hemorrhagic subtype accounts for 15-20%.

The common neuropsychiatric sequelae of stroke include poststroke depression (PSD), poststroke anxiety disorder (PSAD), psychosis, catastrophic reaction, apathy, personality change, aphasia, pathological laughter and crying(PLC) and dementia.

Epidemiology

Poststroke depression is a major cause of morbidity in these patients. PSD may occur acutely in the first few weeks after stroke (in about one-third of patients) or may be delayed even up to 2 years (in about another one-third of patients). One study found 27% rate of major depression and 20% rate of minor depression in the first three months after stroke.

In the poststroke period, anxiety disorder is the second most common psychiatric disorder after PSD. The reported frequencies are as high as 25% in the acute post-stroke period and about 10-20% in the 1-2 years after stroke. More than 50% of patients also fulfill criteria of depression. The early onset PSAD has a mean duration of 1.5 months while late onset PSAD has a mean duration of 3 months.

Patients with poststroke major depression and anxiety disorder have a longer duration than patients with PSD alone.

Etiology

Earlier, PSD was considered to be an 'expected' psychological response of the patient to the stress of serious illness and associated loss of function. Early onset PSD, was considered as a psychological response to physical impairment, compared to delayed onset PSD, which was considered a response to inadequate social functioning. However, PSD appears to be more than just a reaction to loss of function. Psychological factors are undeniably important, but biological factors are also equally important.

The earliest study on PSD found a significantly higher rate of depression in stroke patients compared to orthopedic patients despite similar levels of disability in both, suggesting that these depressive symptoms are more than 'just' a general response to disability. Other studies have also failed to show a consistent relationship between the degree of disability following stroke and the development of PSD.

The disruption of fronto-subcortical circuits, directly or as a distant effect of stroke, plays a central role in causation of depression. The frontal lobes and subcortical nuclei are richly innervated by serotonin and norepinephrine. Anterior and subcortical lesions can interrupt the ascending serotonergic and noradrenergic fibers (from brain stem to frontal lobe, via median forebrain bundle) to a greater extent than posterior and more distally placed lesions, thus resulting in a greater likelihood of PSD. PET studies have shown decreased metabolism in orbito-frontal, anterior cingulate, and inferior temporal regions.

Unlike PSD, the etiopathogenesis of PSAD is not clear, though serotonergic abnormalities are implicated.

Clinical Features

The symptoms of PSD closely follow those of primary depression. However, physical & mental slowness, anxiety, worrying and tearfulness are more common in PSD, while guilt, worthlessness, suicidal ideation, loss of interest and poor concentration are less common, compared to primary major depression.

The cognitive functions are impaired in about 70% of patients with major depression and about 43% in those with minor depression. Aphasia, if present, makes the assessment of both cognitive functions and depression more difficult. Hemispheric lateralization plays a role in the cognitive impairment in patients with PSD. Patients with depression and left hemispheric lesions have a significantly greater cognitive impairment than non-depressed patients with similar lesions. In patients with right hemisphere lesions, cognitive function does not differ between the depressed and non-depressed groups.

The Diagnostic and Statistical Manual of the American Psychiatric Association, IV Edition (DSM-IV) distinguishes between Major Depressive Disorder (primary depression) and Mood Disorder due to a General Medical Condition (Depression secondary to Medical Disorder). PSD would be normally

diagnosed under the category of Mood Disorder due to a General Medical Condition.

Factors Associated with PSD and PSAD

The following factors increase the risk of PSD and PSAD

1. Localization and Lateralization of Stroke.
   a. Bilateral lesions in the anterior frontal and temporal lobes.
   b. Bilateral lesions in caudate nuclei.
   c. Left hemispheric lesions, particularly the anterior frontal lobe and caudate nucleus.
   d. More PSD in left anterior lesions, with severity of PSD directly related to the proximity of the lesion to the frontal pole. In contrast, the severity of PSD in right sided stroke is greater in those with posterior lesions.
   e. The association of PSD with the lesion site is important in acute PSD; with passage of time, this association becomes less marked.
   f. Right sided lesions present with pure anxiety disorder (PSAD) in the absence of depressive symptoms.
   g. Left sided cortical lesions present with PSD along with PSAD, while Left sided sub-cortical lesions present with PSD without anxiety disorder.
2. Older people with micro-vascular disease: Subcortical micro-vascular lesions tend to be greatest in the deep frontal lobe white matter.
3. Gender: Female (Major Depression); Male (Minor Depression).
4. Past history of psychiatric disorder (particularly depression), especially after right-sided stroke.
5. Family history of psychiatric disorder (particularly depression), especially after right-sided stroke.
6. History of prior left-sided stroke, in patients with new right-sided stroke.
7. Negative (stressful) life events in the preceding 6 months.

Assessment

A careful assessment of mood symptoms following stroke can lead to an early diagnosis, institution of effective treatment and reduction of morbidity from PSD.

Pitfalls in the diagnosis of PSD

In stroke patients, the reliance on outward affective expression (sad mood) and general appearance and behavior (emotional lability and bradykinesia) may be unreliable. There may be a mismatch between the patient's (objectively observed) affect and (subjective) mood state.

- PSD patients may deny feeling sad and instead may complain of somatic symptoms.
- The effects of stroke may mimic depression.
- The effects of stroke may impair communication.
- Aphasia may interfere with communication of depressive feelings.

Right-sided stroke can also impair emotional communication (dysprosody) despite intact speech. The dysprosodic patient may appear flat, blunted, and depressed. Therefore it is important to inquire about the patient's underlying mood state.

Injury to the right hemisphere or frontal lobes may cause indifference, or silly or shallow affect, and lack of awareness or minimization of deficit.

Increased emotionality, with brief episodes of tearfulness, is common after stroke. These patients may or may not have a full depressive syndrome.

Increased emotionality, emotional lability and pathological laughter and crying (PLC) may be seen in patients with bilateral frontal lobe or subcortical lesions, with or without pseudobulbar palsy. It is important to inquire about mood state during these emotional outbursts as there is often a disparity between emotional expression and the underlying mood.

Apathetic symptoms (disturbance in motivation) may be really difficult to distinguish from depression. In fact, in some cases apathy may signal depression, while in other cases apathy and decreased motivation occur in the absence of cardinal symptoms of major depression. A patient with apathy loses the ability to initiate routine activities, but may be able to participate in activities initiated by others. A careful assessment of subjective mood state and other core symptoms of depression helps to determine whether apathy is a symptom of depression or not.

Fatigue, loss of energy, poor appetite, and insomnia are common symptoms of depression but these may be caused by the stroke, co-morbid medical conditions, or medications. Again a careful assessment of the subjective mood state and other core symptoms of depression is important in making a correct diagnosis.

Guidelines for the Diagnosis of PSD

To diagnose poststroke depression, the clinician must observe the patient for signs of depression, apathy, and irritability, and inquire about the patient's biological functions like appetite, weight, sleep, and sexual functioning. The amnestic patient may not recall the duration or development of depressive symptoms. Obtaining information from family members and nursing staff is extremely useful. Sometimes, the use of rating scales for depression (e.g. Beck Depression Inventory, Hamilton Rating Scale for Depression, Visual Analog Mood Scale) may be helpful in determining the severity of depressive symptoms and monitoring progress over time.

Course and Prognosis

The majority of patients with poststroke major depression are improved at one year, even without treatment. The mean duration of poststroke major depression is about 39 weeks. Minor depressive symptoms, however, often persist beyond two years. Patients with PSD have lower functional status, increased cognitive impairment, and higher mortality rates than stroke patients without depression.

The presence of depression is associated with a threefold greater mortality rate in the five years following stroke. The mortality rate is highest for depressed patients who are socially isolated.

Anxiety in poststroke period is also associated with high mortality rates, similar to PSD.

Management

Depressive symptoms following stroke are often mild, and resolve without treatment. However, treatment should be initiated for patients with persistent and disabling symptoms. Adequate treatment of PSD improves compliance with rehabilitation.

Pharmacotherapy

There are few controlled trials of the pharmacological treatment of PSD. The antidepressants which have been studied (in controlled trials as well in open studies) include nortriptyline, amitriptyline, trazadone, citalopram, fluoxetine, and bupropion. These have not only been found useful in the management of depression but also in management of pathological laughing and crying (PLC) which sometimes follows stroke.

The general dictum in antidepressant treatment is to 'start low and go slow'. The selection of antidepressant medication is usually guided by safety and tolerability (side effect profile and pharmacokinetic properties), efficacy, and cost factors. Tricyclic antidepressants (TCAs) (e.g. amitriptyline and imipramine) can cause dryness of mouth, constipation, urinary retention, blurring of vision, orthostatic hypotension, sedation, sexual dysfunction, and cardiac conduction disturbances. Presence of narrow angle glaucoma, benign hypertrophy of prostate, or cardiac conduction defect are relative contraindications for the use of TCAs.

The selective serotonin reuptake inhibitors (SSRIs) (e.g. citalopram, sertraline, fluoxetine) are generally better tolerated than TCAs in the elderly depressed population with stroke. The SSRIs can however cause their own side effects, like gastrointestinal symptoms, headache, sexual dysfunction, anxiety and restlessness, and extrapyramidal adverse effects (e.g. parkinsonism, akathisia). SSRIs are highly protein bound and may displace other protein bound medications from their serum binding site, leading to increased serum level of the displaced drug(s). SSRIs have been shown to inhibit a number of hepatic P450 enzymes which may alter the metabolism of other commonly prescribed medications (e.g. beta-blockers, anticonvulsants, some anti-arrhythmic drugs, calcium channel blockers, benzodiazepines, antihistamine medications, and some antibiotics).

Due to frequent comorbidity of PSAD (post-stroke anxiety disorder) with depression, low-dose SSRIs are often the first line of pharmacological management. Low dose, short-acting benzodiazepines and buspirone have also been tried in PSAD. Short-acting benzodiazepines are a sensible choice because longer-acting agents place the patient at a greater risk for adverse effects, such as sedation, ataxias, disinhibition, and confusion.

Stimulants, like methylphenidate and dextro-amphetamine have been also tried in PSD. The improvement in mood is often rapid (within 48 hours) and side effects (like anxiety, anorexia, tachycardia, and insomnia) are usually minimal. Despite the increased risk of seizures after stroke, there were no reports of seizure complicating treatment with stimulants. Low-doses of stimulants often aid in the treatment of apathy or mild depression following stroke; they may also be used to achieve an early and rapid improvement in depressive symptoms while more traditional antidepressant treatments are initiated. However, stimulant use has clinically significant dependence potential and should be judiciously employed.

The data on ECT for PSD is very limited, but ECT may be a useful, relatively safe treatment in severe PSD. The principal contraindications to ECT in PSD are increased intracranial pressure, severe and poorly controlled hypertension, and unstable cardiac rhythm disturbances.

Psychosocial intervention

Although there are no systematic studies of the effectiveness of psychotherapy in the treatment of PSD, psychotherapy is often useful in PSD. Psychotherapy is aimed at helping the patient and family adjust to the loss of function and consequent compromised self-image and self-esteem. The psychotherapy needs to be adapted to the cognitive and language deficits of the patient. The more cognitively impaired the patient, the more behavioral and less verbal the psychotherapy.

Psycho education is important to help the patient and family understand the changes in physical and cognitive function, increase in caregiver responsibilities, and changes in the patient's role within the family. This understanding can improve the family's quality of life after stroke. The psychotherapy along with rehabilitation remains a crucial part of treatment after stroke.

Conclusion

Post stroke depressed patients, who present with persistent and disabling symptoms, should be treated promptly, as apart from reducing the psychiatric morbidity, it also helps to improve the compliance of patient for rehabilitation.

Depression and anxiety after myocardial infarction

Case of post myocardial infarction depression

Mr.Gaitonde a 43 year old, lawyer, has been for the past 2 years, consulting his physician frequently and had undergone repeated medical investigations, as he believed that he was getting repeated episodes of heart attack. These complaints originated 2 years back, when he was admitted to a hospital for chest pain and was diagnosed to have suffered from a myocardial infarction(MI). Since that episode of MI, he became a constant worrier and has always been preoccupied with some degree of tension and apprehension. Since the past 3 months, his symptoms of anxiety and depression have increased in intensity and frequency. He also often experienced, bouts of fearfulness upon waking in the morning, would feel nervous, restless and light-headed and his heart would pound. He had difficulty concentrating on his work and found his attention wandering when arguing for his client in court. He found himself constantly worrying about getting another heart attack and had difficulty in distancing himself from these thoughts. Though, he complained of being afraid to visit the court or to accept stressful cases, he still did not avoid his work or specific situations. His sleep and appetite were normal.

Gaitonde was diagnosed to be suffering from Post Myocardial Infarction Depression(PMID).

Epidemiology

Almost 65% of patients with acute myocardial infarction (MI) report depressive symptoms, with 15-22% suffering from major depression (PMID).

Depression also contributes as an independent risk factor for cardiovascular disease in persons who are otherwise healthy, even after controlling for smoking status, gender, weight, activity, blood pressure and cholesterol levels. Persons, who are depressed and have pre-existing cardiovascular disease, have a 3.5 times greater risk of dying of an MI than patients with cardiac disease who are not depressed, particularly in men.

Clinical features and Diagnosis

Depression is frequently undiagnosed and untreated in patients with heart disease. It is estimated that less than 25% of cardiac patients with major depression are diagnosed with depression, and only about 50% of those patients receive treatment for depression.

The Diagnostic and Statistical Manual of the APA, IV Edition (DSM-IV) distinguishes between Major Depressive Disorder (primary depression) and Mood Disorder due to a General Medical Condition (Depression secondary to Medical Disorder). PMID would be normally diagnosed under this category.

Pitfalls in the diagnosis of PMID

Depression may be under-diagnosed and under-treated in patients with MI because of the following factors

1. Some symptoms (e.g. fatigue, insomnia) are common to depression and coronary artery disease (CAD).
2. It may be erroneously believed that depression is a normal reaction to MI.
3. Patients may be reluctant to report symptoms of depression.
4. Physicians may be reluctant to ask their patients about depression.
5. Physicians may not think of asking about depression, while busy in managing the acute events of MI.
6. Physicians may be reluctant to prescribe antidepressant medications to patients with MI because of potential adverse side effects.
7. Depression and anxiety symptoms are often comorbid.

Factors Associated with Post-MI Depression

Assessment of depression in patients with MI requires an understanding of the risk factors for depression. These include

- Female gender.
- Previous history of depression.
- Family history of depression.
- Lack of social support (especially if living alone) and loss of functioning or major life role.
- Negative (stressful) life events in the preceding 6 months

Management

Assessment

A careful assessment of mood symptoms following myocardial infarction can lead to early diagnosis, effective treatment and reduce morbidity from MI. To diagnose depression, the clinician must observe the patient for signs of depression, apathy, and irritability, and inquire about the patient's biological functions like appetite, weight, sleep, and sexual functioning.

This information should be obtained through a complete interview with the patient and the informants. The use of rating scales for depression (e.g. Beck Depression Inventory, Hamilton Rating Scale for Depression, Zung Self Rating Depression Scale, and Geriatric Depression Scale) may be helpful in determining the severity of depressive symptoms. These instruments are sensitive to changes in depression and, as a result, are also useful for monitoring treatment.

Pharmacotherapy

Antidepressants are indicated if depression is severe, chronic, or recurrent; there is prior history of good response to antidepressants; and psychotherapy is either unavailable or not enough.

The general dictum in antidepressant treatment again is to 'start low and go slow'. The selection of anti-depressant medication is usually guided by safety and tolerability (side effect profile and pharmacokinetic properties), efficacy, and cost. Overall, there is little difference in efficacy among the antidepressants in treating mild to moderate depression. However, there are significant differences in their adverse effect profiles.

As discussed under the treatment of PSD, SSRIs (e.g. citalopram, sertraline) have emerged as the first line drugs in the management of PMID. Unlike TCAs, SSRIs have a negligible effect on the cardiovascular system, even in cases of overdose and lack any pro-arrhythmic or anti-arrhythmic effects. Among newer drugs, venlafaxine may be least desirable, in patients with pre-existing cardiovascular disease, due to possible clinically significant increase in heart rate and blood pressure.

Similarly, TCAs are best avoided as they can have a negative effect on the cardiovascular system (e.g. hypotension). Effects of TCAs on cardiac conduction are similar to that of quinidine, like a class I anti-arrhythmics. The Cardiac Arrhythmia Suppression Trial (CAST) report demonstrated that class I antiarrhythmic therapy caused an increase in mortality rates. Approximately 20% of patients with significant conduction disturbances, such as bundle branch block, exhibit complications while on TCA therapy. Given these side effects and drug interactions, TCAs must be considered to be contraindicated in the setting of an acute coronary event and used very cautiously, if at all, in patients with a history of significant heart disease. Among the other safer antidepressants are tianeptine, and bupropion.

The significance of potential interactions between, antidepressant and cardiovascular medications must be considered when treating depression in cardiac patients. A number of these agents impact the enzyme substrates of the CYP450 system (e.g. fluoxetine, an inhibitor of the CYP2D6, may decrease the rate of metabolism of various beta blockers, thereby leading to bradycardia).

Psychosocial interventions

The most effective psychosocial treatments for post-MI depression are cognitive-behavior therapy (CBT), supportive psychotherapy, assertiveness training, and relaxation techniques. It is prudent to combine the antidepressant treatment with psychosocial management for adequate treatment of PMID. However, psychosocial management is chosen as the only method of treatment if

- Depression is not severe
- Depression is not chronic
- Psychotic features are absent
- Medical contraindication(s) to medications exist
- Recovery has not been achieved with drug treatment alone
- Complex psychosocial circumstances exist

Psycho education addressing the erroneous beliefs (e.g. fear of physical activity, including sexual activity) and expectations about post-MI care is very useful. Identifying these misconceptions and providing supportive education can lead to better adherence to treatment by patients and a more positive approach to their health. Education regarding the illness also improves survival, as it is well known that up to 20% of acute MI events are unrecognized due to denial of MI symptoms, especially in men. Included in this process is encouragement of realistic participation in exercise, hobbies, social activities, and formal rehabilitation programs. Regular use of breathing exercises, relaxation techniques, and/or yoga can help reduce anxiety.

Family members often experience feelings of helplessness, loss and depression similar to those of the patient. It is therefore vital to involve family members in the rehabilitation process of patients with MI.

Conclusion

To achieve adequate response in a PMID patient, a combination of pharmacotherapy (involving the use of antidepressant drugs), & psychosocial interventions should be judiciously used.

Anxiety and Depression associated with Psychosomatic Disorders

Prathap Tharyan

Introduction

Psychosomatic disorders are physical disorders wherein psychological factors influence symptoms and the course of the disorder. The term 'psychosomatic' indicates a mind-body relationship, but current understanding holds that the relative contribution of physical and psychological factors in people with so called psychosomatic disorders vary widely, and that people with disorders not traditionally considered psychosomatic also have varying combinations of physical and psychological contributions to the experience of symptoms, response to treatment, course and outcome.

This chapter deals with functional bowel disorders (particularly irritable bowel syndrome, and non-ulcer dyspepsia), asthma and related respiratory conditions, migraine and other forms of headache, where alleviation of anxiety and depression improves symptom control and attention to psychological stressors influences outcome.

Functional Gastrointestinal Disorders

Case report of recurrent abdominal pain, and diarrhoea

A 35 year old married male, working as a computer programmer for a large business concern, was referred to gastroenterology services by his primary care physician with a history of long-standing abdominal pain, abdominal bloating, and periodic diarrhoea. Antacids and cimetidine had offered little relief. Initially antispasmodics, high fibre diet, and bulking agents (Isaphgul) had helped, but this was short-lived. Anti-diarrhoeal medicines and empirical treatment for giardiasis and amoebiasis had not helped his complaints of frequent loose stools. A gastroenterolgy evaluation did not reveal any abnormality and a diagnosis of irritable bowel syndrome was made. He reported some relief in abdominal pain and diarrhoea when dothiepin 25 mg at night was added to the high fibre diet and bulking agent. However, he continued to be concerned about the possibility of diarrhoea and frequently remained absent from work and reported that he was contemplating quitting his job due to difficulty in meeting assignments because of his abdominal complaints.

A psychiatric referral revealed symptoms of long standing anxiety with episodes of panic when faced with deadlines that he could not meet. He admitted to being a perfectionist and being unable to meet deadlines due to his need to be convinced that he had created a perfect programme. He, however, felt convinced that his bowel symptoms were dietary in origin and had tried many diets with limited success. He reported that when he was on holiday, his symptoms were considerably less and attributed it to dietary changes.

The symptoms reported in this case are non-specific but taken together with the absence of abnormal findings on gastroenterological evaluation, indicate the diagnosis of irritable bowel syndrome(IBS). The differential diagnoses to be considered here include non-ulcer dyspepsia, chronic infective diarrhoea, or Crohn's disease, but the lack of relief with empirical remedies, and lack of evidence of an infective, or other cause of chronic diarrhoea, rules out these disorders.

In psychiatric evaluation, depression needs to be considered, either secondary to the chronic problem and in reaction to difficulty in coping with his symptoms and work, or as a primary diagnosis

with increased help seeking behaviour and excessive somatic concern as a manifestation of depression. However, in this patient, the history of long standing anxiety with periodic increases in performance related anxiety resulting in panic attacks, are features of a generalised anxiety disorder with panic attacks. The lack of core depressive features such as a pervasive low mood, inability to enjoy pleasurable activities, poor appetite, and disturbed sleep rules out a primary depressive syndrome. The symptomatic improvement with dothiepin (a tricyclic antidepressant) is as expected in people with irritable bowel syndrome, but the persistent anxiety and avoidance of meetings leading him to contemplate resignation suggests that a phobic disorder is evolving and that psychological interventions are also urgently required. The patient's attributions are important because while it may be true that certain diets agree with him better than others, the improvement in symptoms when on holiday suggests that reduction in stress could account in large measure to reduced symptoms, rather than or in addition to dietary changes. At any rate, his attributions would need to be addressed and alternate models suggested, if therapeutic efforts are to be better focussed. His perfectionist tendencies that fuelled his anxiety suggest that he has obsessive personality traits (characterised by high expectations and rigid attitudes), and maybe even an obsessive (anankastic) personality disorder. Since he did not complain of distressing intrusive thoughts, or compulsive acts, obsessive compulsive disorder was not a diagnosis that was entertained.

The dose of dothiepin was increased to 75 mg at night with considerable improvement in symptoms and relief from diarrhoea. However, he continued to have anticipatory anxiety about diarrhoea. This was managed in 4 sessions over two weeks where he maintained a diary of his fears and bowel related sensations and gradually learned to dismiss these fears and overcome avoidant behaviours. He was educated about the pathophysiology of IBS and learned to re-attribute the exacerbations in symptoms to stress caused partly by his perfectionist tendencies and worry about not meeting deadlines. He became more realistic about his expectations and this improved his efficiency and contributed to continued symptomatic relief.

Functional gastrointestinal disorders

These are a group of conditions with abnormalities of gastrointestinal function and associated with significant distress wherein no structural abnormalities are evident. They are often associated with high rates of comorbid anxiety and depression, which contribute to worsening of symptoms, treatment seeking behaviour, course, and outcome.

Epidemiology

Earlier studies on the association of psychiatric abnormalities in people with ulcerative colitis (UC) and Crohn's disease (CD) are now considered seriously flawed. However, in a recent study (Simren et al., 2002), 33% of 43 UC patients and 57% of 40 CD patients in remission had IBS-like symptoms. The group with IBS-like symptoms (both UC and CD) had higher levels of anxiety and depression and reduced well-being than those without. Anxiety and depression were independent predictors for IBS-like symptoms in these patients. However-as a group these inflammatory bowel disease patients in remission demonstrated psychological well-being comparable to that of the general population.

It is now acknowledged that while it is important to evaluate for psychosocial factors in anyone with a complex and chronic physical condition, psychological mechanisms for the causation of UC and CD are unproven

Clinical features

Functional gastrointestinal disorders produce symptoms throughout the gastrointestinal tract and include symptoms referable to the esophagus (functional heartburn-acid reflux without anatomical abnormality of the esophagus, functional dysphagia, non-cardiac chest pain), duodenum (functional dyspepsia-epigastric pain mimicking an ulcer, aerophagia- swallowing air and belching), bowel (irritable bowel syndrome, functional abdominal pain), and rectum (functional incontinence, functional anorectal pain, dyschezia difficulty with evacuation).

Functional heartburn is the most frequent gastrointestinal disorder in community surveys, and is present in one third of people surveyed, with functional abdominal bloating (31%), and aerophagia (23%) is also seen frequently.

Irritable bowel syndrome is also common (11.6%), as is functional anorectal pain, but clinic and hospital prevalence estimates for irritable bowel syndrome are higher and possibly reflect greater distress and functional impairment in people affected with IBS, and for people with greater symptom severity more likely to consult doctors. It is estimated that 25-45% of attendees at gastroenterology outpatient clinics have functional gastrointestinal disorders.

Etiology of irritable bowel syndrome

Four mechanisms are involved in the pathogenesis of IBS

- Altered intestinal motility
- Increased visceral sensitivity
- Disturbed intestinal reflexes (intrinsic and extrinsic)
- Psychological disorders

Refinements in the understanding of the physiology of the enteric nervous system (ENS), which controls motility, secretion and sensation, and the close relationship between the central nervous system and the enteric nervous system (the brain-gut axis) aids our comprehension of the pathophysiology of IBS. Visceral hypersensitivity and neurotransmitter imbalance currently receive the most attention as possible mechanisms of IBS. The irritable bowel syndrome (IBS) symptom complex results from altered regulation of gastrointestinal motility and epithelial function, as well as an altered perception of visceral events.

The physiological effects of psychological and physical stressors on gut function and brain-gut interactions are mediated by outputs of the emotional motor system in terms of autonomic, neuroendocrine, attentional and pain modulatory responses.

IBS patients show an enhanced responsiveness of this system manifesting in altered modulation of gastrointestinal motility, secretion, immune function and in alterations in the perceptual and emotional response to visceral events (Mayer et al., 2001; Villanueva et al., 2001).

Psychosocial stressors therefore serve to precipitate exacerbations of symptoms and a vicious cycle ensues where the patient's cognitions and beliefs about disease, lead to behavioural and functional impairment, increased focusing on bodily symptoms, use of health services, iatrogenic disorders and primary or secondary psychiatric disorders (Sharpe & Bass., 1992; Mayou et al., 1995).

Hypothesised psycho-physiological mechanisms in irritable bowel syndrome.

Predisposing factors

Physiological factors:

- Neurotransmitter abnormalities involving 5-HT_3 and 5-HT_4 receptors and opioid receptor systems.
- Alterations in neurotransmitter mediated intestinal motility and secretion.

Psychological factors:

- Personality traits: anxious, under-assertive, excessive health consciousness.
- Illness beliefs, experiences and attitudes.

↓↑

Precipitating factors

- Life events and stressors
- Dietary factors
- Psychiatric disorders
- Illness beliefs and fears

↓↑

Maintaining factors

- Visceral hypersensitivity
- Attributions, illness beliefs, and fears
- Iatrogenic factors
- Abnormal illness behaviour
- Autonomic arousal
- Excessive focussing on bodily symptoms
- Loss of control and coping mechanisms
- Reactions of others
- Sick role
- Functional and behavioural impairment
- Psychiatric disorders

Peptic ulcer, ulcerative colitis, and Crohn's disease

These disorders were traditionally considered psychosomatic disorders, but current concepts acknowledge that physical factors are more important though, in the individual patient, psychological factors may play an important role in the clinical expression of symptoms, and secondary anxiety and depression may coexist in people with these disorders.

The role of H. pylori in the etiology of the majority of gastric and duodenal ulcers, and the dramatic and enduring response to appropriate antibiotic therapy, has replaced belief that stress mediated increase in gastric acid secretion is the most important etiological factor. What is unclear is why the majority of people with H. pylori infections do not develop ulcers. Speculations include stress related reductions in immune response resulting in higher vulnerability to H. pylori infection, and psychosocial factors mediating the expression of symptoms.

Management of functional gastrointestinal disorders

Irritable bowel syndrome

Pharmacotherapy

Specific treatment depends on whether diarrhoea or constipation is predominant. Prokinetic drugs like cisapride reduce symptoms in constipation predominant patients while actually worsening symptoms in diarrhoea predominant patients (Noor et al., 1998). Emerging therapies for IBS using 5-HT mediation include, 5-HT_3 antagonists, such as ondasetron, granisetron and alosetron, as well as 5-HT_4 agonists such as tegaserod and prucalopride. In addition to opioid agonists (e.g. fedotozine), several other drugs that act on other ENS receptors are being studied, though some of these drugs (alosetron, tegaserod) have toxicities that limit their utility, and the modest improvements in randomised trials seem limited to women (Talley, 2001).

Tricyclic antidepressants (especially amitriptyline and dothiepin) in doses ranging from 25-75 mg/day are useful in reducing visceral hypersensitivity and diarrhoea in diarrhoea predominant patients and can be combined with bulking agents in constipation predominant patients. This response is independent of the presence of depression or anxiety (Rajagopalan et al., 1998), though if depressive symptoms are prominent, the dose of the tricyclic should be increased to 150 mg/day. Antidepressants are effective in the treatment of generalised anxiety and while all classes of antidepressants are effective, the evidence is best for tricyclics, trazadone, and venlafaxine (Kapczinski et al., 2002). The specific serotonin reuptake inhibitors (SSRI) such as fluoxetine, and sertraline, are also effective anxiolytics, and produce little drowsiness and no anticholinergic side effects, but due to their tendency to produce nausea and loose stools in people with IBS, their use may be limited to those with constipation predominant disorder in whom tricyclics may worsen constipation.

Psychosocial intervention

Multicomponent behaviour therapy includes providing IBS information and education, progressive muscle relaxation, training in illness-related cognitive coping strategies, problem-solving, and assertiveness training, and when added to standard medication in 10 sessions over 10 weeks was found to be superior in reducing IBS symptoms and overall well-being, than medication alone (Heymann-Monnikes et al., 2000). Relaxation training, hypnosis, cognitive therapy, meditation, and

stress reduction have all been used in addition to medication in alleviating symptoms in people with IBS.

The majority of patients with IBS do well with these interventions, though around 15% show persistent symptoms. Brief psychodynamic psychotherapy aimed at establishing the link between emotional distress stemming from psychological factors in their past or current relationships, and their current physical symptoms, has been found to be beneficial in those people with refractory IBS who are able to acknowledge emotional symptoms, are open and willing to discuss these, and work at resolution of these feelings. Those who did not have these characteristics or had chronic, unremitting pain did poorly with psychotherapy (Guthrie et al., 1993).

Non-ulcer dyspepsia

Tricyclic antidepressants are also useful in the treatment of non-ulcer dyspepsia along with life style and dietary changes, and proton pump inhibitors or prokinetic drugs (Paganamamula et al., 2002). Psychological interventions in the form of psychodynamic psychotherapy and cognitive therapy are also useful for people with non-ulcer dyspepsia (Soo et al., 2002). Brief psychodynamic psychotherapy focussed on interpersonal issues was found to be superior to supportive listening in a randomised trial of people with functional dyspepsia resistant to medical treatment, with significant beneficial effects lasting up to one year (Hamilton et al., 2000).

Asthma and Related Respiratory Disorders

Case report of a young woman with severe breathing difficulty

A thirty year old woman presented to casualty services with severe breathing difficulty of several hours duration. She had been looking after her demented mother for several months and had not been able to attend work regularly with resultant threats of losing her job. The mothers illness and her involvement in caring for her mother had exacerbated marital problems, and she had been anxious and depressed and had entertained thoughts of suicide. She had experienced episodes of asthma in childhood but had been symptom free for many years until the present episode. After her condition had stabilised with bronchodilators and nebulisers, she was referred for psychiatric evaluation. She and her husband were seen in subsequent sessions and problem solving techniques were used to generate possible solutions, which in this case involved transferring the patient's mother to her son's house temporarily until hired help could be found to help take care of the mother. During follow-up sessions, marital issues were also addressed. At one-year follow up she was free of respiratory problems and marital functioning was considerably better, even though her mother was living with her and hired help was only intermittently available.

Asthma is characterized by airway hyper-responsiveness, inflammation, and reversible obstruction. Respiratory tract infection, allergies, air pollution, and psychosocial factors influence the severity and frequency of asthma symptoms. In the above case, the acute attack of asthma was triggered by exhaustion, and stress, which was identified by medical staff in casualty and appropriate intervention organised.

The psychiatric diagnosis made was adjustment problems presenting with mixed features of anxiety and depression, but not severe enough to warrant specific psychotropic medication. Here problem solving and marital counselling were sufficient to mitigate stressors and reduce symptoms in conjunction with inhaled bronchodilators.

Epidemiology

The prevalence of panic disorder and agoraphobia is higher among asthmatics than in the general population, and the presence of anxiety disorders worsens the prognosis of asthma and often increases presentation to emergency care (Rietveld et al., 1999).

Etiology and Clinical features

Role of psychological factors in asthma

Psychological factors can affect the expression of asthmatic attacks in several ways. Chief among these are perceptions and reactions to changes in airway resistance, and comorbidity with anxiety and panic disorders. There are wide variations in perception of changes in airway resistance. Some people are especially sensitive to these changes and this may be useful in initiating appropriate and timely medical treatment, or may lead to increased anxiety, which may worsen asthma. Conversely, some people are less likely to perceive increases in airway resistance; this is particularly so in the elderly. This leads to poorer, and sometimes fatal, outcomes.

Emotional arousal mediated by brainstem mediated sensitivity to elevated Pco_2 may provoke increased firing from the locus ceruleus, resulting in panic attacks. Patients with comorbid asthma and panic disorder have greater anticipatory anxiety about panic attacks and acute asthmatic exacerbations and this anxiety may trigger both types of attacks. It may be necessary to use objective measures of lung function such as peak flow meters during attacks to disentangle hyperventilation from asthma.

It is important to remember that the side effects of anti-asthma drugs can produce symptoms mimicking anxiety, such as tremor, agitation, and insomnia. These may indicate the need to review the doses of medication and only rarely require antianxiety measures; Beta-adrenergic blockers such as propranolol are contraindicated in such instances due to their propensity to worsen asthma. In addition, steroids used to treat asthma can cause agitation, euphoria, and depression, though this is usually dose dependent and rarely seen in low doses in the absence of a personal or family history of mood symptoms.

Apart from anxiety and panic, depression is often a result of poorly controlled asthma and one consequence is decreased adherence to medication. Depressed people often have comorbid anxiety symptoms, which often resolve when the depression lifts. Children with asthma are at special risk for problems in psychological functioning, as are children with other chronic illnesses. Children in a negative emotional state, uncertain about the condition of their airways, are inclined to interpret exercise-related general sensations (fatigue, heart pounding, sighing) as symptoms of airways obstruction. Consequently, they may report relatively high breathlessness, irrespective of actual objective symptoms of asthma (Rietveld et al., 1998).

Differential diagnosis

The other conditions to be considered are panic attacks with hyperventilation, and vocal cord dysfunction. Asthma sufferers, especially high frequency attendees at emergency rooms, report more panic anxiety symptoms, and people with hyperventilation due to a panic attack are sometimes mistaken to have an acute asthmatic attack.

Vocal cord dysfunction (VCD) is a disorder caused by abnormal vocal cord movements that is often

confused with asthma. Patients with this disorder are often treated for refractory asthma as the laryngeal sounds produced by this disorder resemble wheeze or stridor. Adduction of the anterior two thirds of the vocal cords on laryngoscopy confirms the diagnosis, which should be entertained in anyone with refractory asthma, especially if stridor is noticed. People with vocal cord dysfunction often have a higher prevalence of anxiety disorders (Gavin et al., 1998). Speech therapy is often useful in treatment of the dysfunction, and antianxiety measures are useful in people with comorbid anxiety.

Management of anxiety and depression associated with asthma

Pharmacotherapy

The medical management of asthma with anti-asthmatic medication should be complemented by an assessment for comorbid anxiety, panic and agoraphobia, and depression, and the possible contribution of medication in symptom production. The SSRI class of antidepressants are specially indicated (along with Trazodone 25-50 mg at night for sleep, if needed) as they are well tolerated and are less likely to add to side effects of concurrent medication.

Psychosocial intervention

There is some indication that family therapy may be a useful adjunct to medication for children with asthma (Panton & Barley, 2002). Education about symptoms and medication are an important component of management and in anxious non-steroid dependent patients, relaxation training and biofeedback reduce asthma severity and use of bronchodilators.

Migraine and other causes of Headache

Case report of a young woman with intense headache

A psychiatric consultation was sought for a 28 year old woman who had consumed 30 tablets of 25 mg amitriptyline, which had been prescribed for chronic headache. She revealed that she had taken the tablets because she was unable to tolerate the intense headaches she had been suffering for the previous 10 years. She had planned the overdose, had harboured a clear intent to die, and had taken measures (ultimately inadequate) to escape detection of the overdose. Further enquiry revealed that she worked as a teacher in a remote hilly area among tribal people and, though she enjoyed her work, she found the journey back home on weekends tiring. Furthermore, she ended up cooking and cleaning for her husband, three children, and mother-in-law and resented the fact that they let dirty dishes and clothes accumulate for her return. Her husband was overtly emotionally supportive but, in her opinion, covertly critical of her and concerned more about the welfare of his mother. She felt lonely during the week but felt used by her family over the weekends. She also resented her husband's sexual overtures every weekend and often pleaded a headache to escape physical intimacy since emotional intimacy was not forthcoming. Her mood was low and she complained of insomnia with early awakening over the previous three months. She admitted to a worsening of her headaches over the same period and admitted that the headaches were reasonably controlled before that.

Evaluation of her headaches revealed a previous pattern of right unilateral pulsating headaches accompanied by intolerance to light and noise, nausea and vomiting, and without an aura. The intensity invariably worsened with exertion and usually lasted 24-48 hours. In recent months, the pattern had changed to bilateral, constant, vice-like pain often lasting a week, but she denied nausea or vomiting and physical exertion left her more tired than usual. without worsening of the headache

She was treated with a combination of sertaline 50 mg/day in the daytime and trazodone 50 mg at night with which her mood and sleep improved within 10 days. When she was better, she was seen with her husband for three sessions of psychodynamic-interpersonal psychotherapy wherein she expressed her difficulties, anger, and resentment. Her husband expressed his bitterness at her refusal to cooperate for sexual relationships and his belief that she sometimes appeared to be feigning symptoms. Communication strategies within the marital unit were discussed and the patient developed ways in which she communicated her distress directly rather than through somatic symptoms or through deliberate self-harm. She was seen monthly for three months and at six monthly intervals over two years. At two year follow-up, she remained mostly free of headaches, and the marital relations were satisfactory for both partners.

The most common causes of primary headache are migraine, tension-type headache, and cluster headaches, though frequently migraine and tension headaches may coexist, often simultaneously or alternate over time. Cluster headaches tend to cluster in time with long symptom free intervals, and the headache is unilateral, orbital, supraorbital, or temporal with autonomic changes in the eye (lacrimation, conjunctival injection, myosis, ptosis, eyelid oedema), or nose (nasal congestion, rhinorrhoea) on the same side.

The description of this woman's long-standing headache is typical of migraine without an aura, though the subsequent headaches are descriptive of tension headaches. Some evidence of 'abnormal illness behaviour' where she exaggerated her symptoms to gain sympathy, and use of the 'sick role' to avoid unpleasant events was also present. In addition, she had developed a depressive episode with some biological disturbances that had worsened her headache and probably reduced her pain threshold. Treatment warranted an adequate dose of an antidepressant. The dose of amitriptyline could have been increased to 150 mg/day to effectively treat depression and control headaches, but the potential for a further overdose necessitated the use of drugs that would be relatively safer. SSRI's such as sertraline are safe in overdose and, in this case, proved effective, though in some people, headache and nausea may be reported, especially at initiation. Trazodone, in doses of 25-50 mg at night, is also safe and an effective hypnotic. The combination is usually well tolerated, though infrequently the combination may result in a serotoninergic syndrome. Here psychotherapy focussed on interpersonal issues proved effective in alleviating her distress.

Epidemiology of migraine and other primary headache syndromes

Headaches affect 60-80 %of the general population, but lifetime prevalence for migraine ranges from 4-19% among men and 8-29% for women (Stewart & Schechter, 1994). It is estimated that regular activities are limited during 78% of migraine attacks and 38% of tension-type headaches (Edmeads et al., 1993). The peak incidence of migraine is between the ages 20-45 year, but may begin in childhood. It is more common in women, increases during puberty and pregnancy and with hormone therapy, and decreases after the menopause. A family history of migraine is often, but not invariably, present. People with migraine often suffer from asthma, allergies, and cardiovascular diseases.

Psychiatric comorbidity is common and mood disorders, (including bipolar affective disorder), anxiety disorders, panic disorder and phobias are frequently encountered.

Cluster headaches are relatively rare and are mostly confined to males. Tension headaches occur more commonly than migraine, are also seen more frequently in women and young adults, but can be

seen in people of all ages. First degree relatives of people with chronic tension type headaches (CTTH) have about 2 to 4-fold significantly increased risk of CTTH compared with the general population (Russel et al., 1999).

Etiology

Migraine is believed to occur due to a series of neurochemical, neurovascular and neuro-inflammatory changes triggered by environmental events, internal mood and physiological states. Evidence has accumulated that the prodromes of migraine are due to cerebral vasoconstriction and headaches of both cluster and migraine are due to painful dilatation. Theories regarding their pathogenesis include cyclic release of vasoactive substances and other neurotransmitters (such as serotonin, catecholamines, histamine, acetylcholine, prostaglandins, substance P, endogenous opiates) from platelet and/or other sources. These substances influence vasomotor receptors bringing about abnormal constriction and/or dilatation.

Regarding tension headaches, the earlier belief that sustained muscle contraction caused by stress was responsible, has not been borne out by investigations, as treatment with botulinum toxin, normally effective in treating muscle tenderness and pain, was ineffective in relieving pain in tension headaches (Rollnick et al., 2000). The current belief is that tension headaches, like migraine, involve serotonin activation and neurovascular mechanisms.

In cluster headaches, pain is associated with vasoconstriction of the internal carotids and there is presumptive evidence that the decrease in blood flow and increase in vascular resistance may be due to constriction of intracranial arteries by reflex activation of sympathetic efferents, rather than to decrease of arterial CO2 tension (Hannerz & Jogestrand, 1993).

Management of migraine and associated headache syndromes

The evaluation of headache involves the exclusion of vascular malformations, structural lesions, infective causes, intracranial abscess or haemorrhage, metabolic conditions, brain tumours, and disorders of the sinuses, teeth, jaws, eyes, and spine.

Pharmacotherapy of Migraine

Treatment of migraine can be abortive (taken at the onset of the episode), palliative (taken after the episode has begun), or prophylactic, with daily medication to prevent attacks.

For acute attacks, symptom relief is often provided by NSAIDs, and analgesics, ergot derivatives, and opioids. Sumatriptan, a selective 5-HT_{ID} agonist available as parenteral injection, oral tablets, or nasal. Spray, is effective in providing almost instantaneous relief of migraine, as is Zolmitriptan. Sumatriptan was found to be significantly superior to placebo in relieving migraine, migrainous, and episodic tension-type headache in people with migraine (Lipton et al., 2000), with effects lasting upto 2 hours post dose.

In prophylaxis of migraine, many classes of drugs are effective and the choice of drug would depend on comorbid conditions, patient's tolerance, and preference. Evidence is strong for the prophylactic efficacy of beta-blockers and tricyclic antidepressants (Ziegler et al., 1987). Metoprolol is preferable to propranolol in people with migraine and asthma and beta-blockers are more effective in people with migraine and high levels of autonomic anxiety.

Tricyclic antidepressants are effective in migraine prophylaxis even in the absence of significant depression. Amitriptyline has been the best studied in migraine prophylaxis but other tricyclics such as dothiepin are as effective in clinical practice and may be better tolerated. Effective doses range from 75-150 mg/day.

SSRIs have not been sufficiently investigated, and may not always be useful in migraine but may be more effective in mixed pain syndromes. At any rate they should not be given along with sumatriptan to prevent serious drug interactions.

Other agents effective in prevention of migraine include nifedipine (Shukla et al., 1995) and other calcium channel blockers, NSAIDs, and sodium valproate (especially useful in those with comorbid bipolar affective disorder).

Psychosocial intervention

Non-pharmacological treatments effective in prophylaxis include relaxation training and stress-coping training with beneficial effects maintained over 3 year follow-up. Thermal biofeedback and interventions combining biofeedback and progressive muscle relaxation are superior to either alone.

Pharmacotherapy of Tension type Headaches

Analgesics and NSAIDs are the first line treatment for acute tension type headaches. Sumatriptan is not particularly effective. The treatment of chronic tension type headaches are similar to that used in prophylaxis of migraine, and antidepressant medication and stress management therapy are each modestly effective in treating chronic tension-type headaches. Combined therapy may improve outcome relative to monotherapy (Holroyd et al., 2001).

Tricyclics such as amitriptyline elicits its analgesic effect in chronic tension type headaches by reducing the transmission of painful stimuli from myofascial tissues rather than by reducing overall pain sensitivity, suggesting that this effect is caused by a segmental reduction of central sensitisation, in combination with a peripheral anti-nociceptive action (Bendtsen & Jensen, 2000).

Buspirone may also be effective in chronic tension type headache though not as effective as amitriptyline. SSRI's are not always effective in treating chronic tension type headaches in the absence of a concomitant depression (Bendtsen & Jensen, 2000), though in individual cases, they may provide relief.

Alprazolam has also been shown to be effective in the short-term prophylaxis of chronic tension headaches (Shukla et al., 1996), though the long-term effects are unknown.

Psychosocial interventions

Relaxation training, biofeedback-assisted or otherwise, appears to be an efficacious in prophylaxis with durable effects.

Pharmacotherapy of Cluster Headaches

The acute attacks respond instantaneously to hyperbaric oxygen, but due to lack of availability, parenteral sumatriptan or injected opioids are the common effective strategies. Oral zolmitriptan is also effective in aborting attacks.

Antidepressants are not effective in prevention, though lithium and sodium valproate are, as are calcium channel blockers.

General principles in the management of people with psychosomatic and functional somatic disorders

The following principles apply to the treatment of people with any of the psychosomatic disorders discussed above, as well as to those who present with unexplained somatic symptoms. In both contexts, the physician-patient relationship is of crucial importance and adherence to simple principles increases the chances of a positive outcome for both parties. Most patients with the above problems can, and should, be treated by primary care physicians, with referral to specialist services only in refractory conditions. Even then, it is best to provide coordinated care.

Assessment

1. The aims of assessment are to clarify the nature of the patient's complaints, understand what he/she particularly wants, elicit the patients fears and beliefs about the illness, exclude organic disease, identify emotional distress and disorder, and to identify relevant psychological and social stressors.
2. A clinical diagnosis needs to be supplemented with an etiological formulation which could include what one has gleaned about possible predisposing, precipitating, and maintaining factors, ordered according to biological, psychological, and social domains. Following table provides such a formulation for the lady in the case report with migraine.

Etiological formulation of a young woman with migraine and a suicidal attempt.

	Predisposing	Precipitating	Perpetuating
Biological	Mother had migraine Sensitive temperament	Exhaustion	Excessive NSAID use Tension headache
Psychological	Expects support Long standing headache	Demand for sex No emotional support	Depression Continuing headache
Social	Marital problems Loneliness	Quarrel Long travel	Sick role, illness behaviour Mother- in-law's reaction

Management

1. Make the patient feel understood. Listen to the patient's story and validate the patient's reactions. Do not be dismissive, by stating that 'it's all in your mind', or by offering premature reassurance.
2. Establish a collaborative relationship. Do not offer to 'cure' the patient but offer to work together to make things easier for the patient within a realistic period.
3. Elicit the patient's explanatory model for the problem, fears, and misconceptions. Do not just say there is nothing wrong, or that the problem is minor, but offer an explanation for the actual cause of their symptoms, incorporating aspects of the formulation, if possible. Do not invent reasons for which there is no empirical evidence.

4. Use language that a patient would understand. Use examples or analogies and/or diagrams to illustrate your point.
5. Avoid unnecessary investigations, but check to see if patient is satisfied that investigations done have been thorough enough and the results have been properly understood.
6. Incorporate life style changes that would reduce stress, resolve conflict, and improve physical fitness (diet, activity, avoiding substance abuse). Help the patient identify sources of stress and links with exacerbations or onset of symptoms.
7. Reattribution: This is the process of developing alternative explanations of symptoms in a collaborative manner, using behavioural experiments (for example, getting the patient described above with IBS to go on holiday while trying different diets to see if the lack of stress is sufficient to reduce symptoms).
8. Expose the patient to situations he/she avoids. Prevent focusing on symptoms, or seeking reassurance. Identify inappropriate reassurance seeking and educate the patient of the need to cease doing so. Provide only new information or re-phrase information in ways that are better understood by the patient.
9. Involve the family as co-therapists. Relatives often have misconceptions that reinforce patients' beliefs. These need to be identified and corrected. Relatives can then help motivate patients to follow advice regarding diet, ensure compliance with medication and other advice and most importantly, not sabotage treatment.
10. **Drug treatments:**

- **The use of benzodiazepines:** Benzodiazepines are over prescribed in primary and secondary care and both lorazepam and alprazolam are more dependence producing than the longer acting diazepam. Antidepressants are effective anxiolytics with far less abuse potential. Anxiety and depression often co-exist and antidepressants are more effective in these situations than benzodiazepines alone. In depressed people it may be justifiable to use a combination of a benzodiazepine and antidepressant in the early stages as both symptomatic improvement and lower drop out rates may justify the use of this combination, but the potential benefits must be balanced judiciously against possible harms including development of dependence and accident proneness.
- **The use of antidepressants:** It is better to over prescribe antidepressants than to not prescribe them at all. Used judiciously, they afford symptom improvement even in the absence of depression or anxiety as they have multiple sites of action; their name as antidepressants is perhaps too limiting. There is also evidence that substantial benefit can accrue from the use of antidepressants for the treatment of multiple unexplained symptoms (O'Malley et al., 1999).

Conclusion

When managing a patient with psychosomatic & functional somatic disorders, one should not expect a cure but gradual improvement over longer periods. Therapeutic interventions in these patients bring about small changes that will be useful to the patient, help arrest deterioration, reduce distress, and help them lead fuller lives.

Anxiety and Depression in Terminally ill Patients

Ashu R.Gandhi
Santosh K.Chaturvedi

'The functions of medicine are threefold: to relieve pain, to reduce the violence of disease, and to refrain from trying to cure those whom disease has conquered, acknowledging that in such cases medicine is powerless'.

Hippocrates

case of sarcoma

Mr ravi, a 65 year old male reported complaints of sleep disturbance and multiple aches and pains during routine hospice rounds. He had been diagnosed to be having soft tissue sarcoma of right thigh and had undergone surgery as well as radiotherapy with no substantial improvement and there was rapid progression in the size of the tumor. Because of poor financial status and poor support from family members he could not undergo further active management and was thus in the hospice for symptomatic management.

A mental status examination revealed pervasive feelings of tension as well as sadness, inability to enjoy usual activities, decreased appetite, fatigue, no interest in day to day activities like shaving, bathing, etc; tearfulness while talking to the staff, feelings of having slowed down, and sudden feelings of panic. A further careful clinical interview revealed significant global ideas of hopelessness, helplessness about the physical condition, death wishes, worrying most of the time, and inability to look ahead with enjoyment to things. He also revealed that he did not feel that he was getting adequate support from his family and felt abandoned by them.

In view of the features of both anxiety and depression due to his disease and circumstances, a diagnosis of, adjustment disorder with mixed anxiety and depressed mood was made. He was started on a low dose of tricyclics (amitriptyline 25 mg at bedtime), which was titrated upwards and an effort was made to explore his concerns, help him ventilate and counsel him about living in the present. Although some improvement in sleep, tension and depression was noticed, there was steady deterioration in his physical status and the patient expired two weeks later.

These symptoms of fatigue, feelings of having slowed down, decreased appetite, and inability in performing daily activities could be due to the rapidly progressive sarcoma, or due to depression or anxiety or both. So, how does one diagnose depression in a person with terminal illness? Although various approaches have been suggested, endicott's substitution criteria, which are considered to be the most helpful to diagnose depression according to dsm-iv (apa, 1994) are discussed below.

The other questions that are raised are that what is the psychiatric morbidity in terminally ill patients? How to recognize it? Why should one treat psychiatric illness in a terminally ill patient and how should we treat and manage it? Are the issues different in a terminally ill patient? The answers to some of these questions are discussed in the following account.

Depressive disorders in terminally ill

Though depression is very common in the setting of terminal illness, studies show that it continues to be under diagnosed and under treated in the terminally ill patient population (davidson & meltzer-brody, 1999; goldberg & mor, 1985). Depression becomes more prevalent and severe as comorbid physical illness progresses (rodin et al., 1991).

Epidemiology

Prevalence rates for major depressive disorder in patients with cancer range from 4.5-58% (levine et al.,1978; massie & holland, 1987; massie et al.,1979). Depression is more common in the advanced stages of the disease, ranging in prevalence from 23-58% (bukberg & holland, 1980; chochinov et al., 1994). Derogatis et al., (1987) reported that slightly more than half (53%) of the 215 terminally ill patients evaluated, were adjusting normally to stress; the remainder (47%) had clinically apparent psychiatric disorders. Of this 47% with psychiatric disorders, more than two-thirds (68%) had reactive anxiety and depression (adjustment disorders with depressed or anxious mood); 13% had a major depression; 8% had an organic mental disorder; 7% had a personality disorder; and 4% had a preexisting anxiety disorder. As noted above in this study it was seen that nearly 90% of the psychiatric disorders observed were reactions to or manifestations of disease or treatment.

According to the nimhans-kimio study (1991-1998), which assessed psychiatric morbidity in 300 mixed diagnosis cancer patients; around 53% of the patients had significant psychiatric morbidity. Overall, the most common diagnosis was depressive disorders (22%), followed by sleep disorders (15%), adjustment disorders (9%), mixed anxiety and depression (6%), and anxiety disorders (1%).

Etiology

Depressive symptoms in advanced cancer patients can be due to various factors, as follows:

1) the disease itself.
2) symptoms of the disease, like pain.
3) the psychosocial impact of cancer.
4) consequence of a paraneoplastic syndrome of the cancer.
5) side effect of the treatment like radiotherapy, surgery, or chemotherapeutic regimens.
6) comorbid disorders.

It needs to be emphasized that depressed mood and sadness can be appropriate responses as the terminally ill patient faces impending death, and these emotions can be manifestations of anticipatory grief over the impending loss of one's life, health, loved ones and autonomy. In a clinical situation depressive symptoms may present either independently or as a part of psychiatric syndromes.

Risk factors for depression in terminally ill

Cancer patients who are at higher risk for depression include those with poor physical condition, inadequately controlled pain, advanced stage of illness and preexisting mood disorders; those with pancreatic, lung, or head and neck cancers; and those with significant life stresses or recent losses (massie & popkin, 1998). Treatment with some commonly prescribed medications, such as methyldopa, propranolol or diazepam, as well as chemotherapeutic agents like vincristine, procarbazine, asparginase and interferon, may also produce symptoms of depression.

Clinical features

The diagnosis of a major depressive disorder in a patient who is terminally ill can be problematic, since terminal illness itself can produce many of the physical symptoms that are characteristic of

major depression in the physically healthy population (endicott, 1983; massie & holland, 1990; plumb & Holland, 1977). Essentially, four approaches to the diagnosis of major depression in the cancer patient have been described in the literature (mcdaniel & nemeroff, 1993), endicott's substitution criteria have found more acceptance with various researchers (endicott, 1983). Despite various approaches, clinical wisdom and experience suggest that greater emphasis should be focused on psychological symptoms like pervasively depressed mood, loss of interest, hopelessness, helplessness, excessive guilt, feelings of worthlessness and desire for death rather than physical symptoms. A simple question, 'are you depressed?' is one of the most accurate and reliable screens for depression of clinical significance in terminally ill patients (chochinov et. Al., 1997). Pervasive hopelessness that is accompanied by a sense of despair or despondency is more likely to represent a symptom of a depressive disorder (massie and holland, 1990). It should be noted that suicidal ideation, even if mild and passive, is very likely to be associated with a significant degree of depression in terminally ill cancer patients (breitbart, 1987).

endicott substitution criteria
For diagnosis of depression (endicott,1983)

Physical/somatic Symptoms	**psychological substitute symptoms**
1. Change in appetite/weight	1. Tearfulness, depressed appearance
2. Sleep disturbance	2. Social withdrawal, decreased talkativeness
3. Fatigue, loss of energy	3. Brooding, self pity
4. Diminished ability to think/concentrate, indecisiveness	4. Lack of reactivity

Course and prognosis

Depression clearly diminishes the quality of life of a patient (wells et al., 1989), and is also a determining factor in the desire to die in many patients who opt for assisted death (chochinov et al., 1995). Consequently depression is a problem of major clinical importance in the care of terminally ill patients and should be a priority in routine assessment. In the case vignette described above, there were significant death wishes that can be understood to be due to depressive disorder.

Management of depression in terminally ill

Evaluation of cancer-related organic factors that can present as depression must precede initiation of treatment. These factors include administration of corticosteroids, chemotherapeutic agents, whole brain radiation, central nervous system metabolic and endocrine complications (breitbart, 1989) and paraneoplastic syndromes (patchell & posner, 1989).

Depression in patients with advanced disease is optimally managed with a combination of supportive psychotherapy, cognitive-behavioral techniques, and antidepressant medications (massie & holland, 1990)

Pharmacotherapy

Psychopharmacological interventions are the mainstay of treatment of terminally ill patients with moderate or severe depressive symptoms who meet criteria for a major depressive episode (massie & holland, 1990). The efficacy of antidepressants in the treatment of depression in patients with cancer has been well established (massie & holland, 1990).

Choice of pharmacotherapeutic intervention

Factors such as prognosis and the time frame of treatment may play important roles in determining the type of pharmacotherapy that is chosen. Tricyclics can improve sleep even on initiation of therapy and thus consequently also affect the anxiety symptoms and distress associated with lack of sleep (chaturvedi et al., 1994). Patients who are within hours to days of death and in distress are likely to benefit most from the use of sedatives or narcotic analgesic infusions.

Treatment with tricyclic antidepressants (tcas) should be initiated at low doses (10-25 mg at bedtime), especially in patients who are debilitated and have advanced disease. The dose should be increased slowly, in increments of 10-25 mg every 1-2 days until a beneficial effect is achieved. Patients with cancer who have depression often have a therapeutic response at much lower doses (25-125 mg/day) than are usually required in patients who do not have medical illness (150-300 mg/day) (massie & holland, 1990). Sedating tricyclics such as amitriptyline or doxepin may be prescribed for the patients with agitation and depression accompanied by insomnia (chaturvedi and chandra, 1998). Desipramine or nortriptyline have relatively low anticholinergic potential and so are useful when one must avoid urinary retention, decreased intestinal motility, or stomatitis. Tricyclics such as amitriptyline and imipramine are preferred in patients with depression associated with pain, as they have analgesic effect and can benefit both symptoms and also act as an adjuvant to narcotic analgesics (chaturvedi and chandra, 1998).

The specific serotonin reuptake inhibitors (ssris) are agents without any anticholinergic effect and have a comparatively better side effect profile than tricyclics and maois. Amongst the ssris sertraline and paroxetine have been useful additions to the list of choices of antidepressants for patients who are terminally ill. Paroxetine may have the additional benefit of being a potent analgesic agent for the management of neuropathic pain (sindrup et al., 1990). A cautious use of fluoxetine may be advocated in the terminally ill patient in view of reported side effects like mild nausea, a brief period of increased anxiety, appetite suppression that may last for several weeks, serious interactions with procarbazine (a chemotherapeutic agent), as well as risk of causing hyponatremia in patients taking antibiotics (mermelstein and lesko, 1992).

Psychotherapy

Supportive psychotherapy and counseling are useful treatment approaches to depression in the patient who is terminally ill. Counseling with the dying patient consists of active listening with supportive verbal interventions and occasional interpretation (cassem, 1987). Despite the seriousness of the patient's plight, it is not necessary for the psychiatrist or psychologist to appear overly solemn or emotionally restrained. Often, it is only the counselor, of all the patient's caregivers,

who is comfortable enough to converse light heartedly and allow the patient to talk about his or her life and experiences rather than focus solely on impending death. The dying patient who wishes to talk or ask questions about death should be allowed to do so freely, with the therapist maintaining an interested, interactive stance.

Anxiety disorders in terminally ill

Anxiety, at least at some level, is probably universal at the end of life. Anxiety accompanies nearly all phases of dying but may take very different forms. This fact makes anxiety difficult to evaluate in the setting of terminal illness and probably contributes to under recognition of anxiety disorders in the dying (shuster et. Al., 1998). Anxiety and anxiety disorders can cause a great deal of suffering in dying patients, especially when anxiety complicates respiratory distress (rosenbaum et. Al., 1997). Anxiety symptoms can cause substantial suffering and are also focus of palliative intervention. Anxiety that persists and causes impairment is considered pathological. Anxiety presents a barrier to relaxation and attention to important relationships, achievement of life goals, and integration of the experience of dying.

Epidemiology

Studies have shown that anxiety may be observed in 70% of terminally ill (chochinov, 2000). Rates of anxiety at the end of life increase with the presence and severity of concomitant physical illness.

Etiology

Presence of anxiety symptoms in a terminally ill patient should make the physician and psychiatrist consider various possibilities. When faced with a terminal illness, patients with preexisting anxiety disorders are at risk for reactivation of symptoms. A generalized anxiety disorder or panic disorder is apt to recur, especially in the presence of shortness of breath or pain. Posttraumatic stress disorder may be activated in dying patients as they relate their situation to some prior near-death experience and the terror associated with it.

On the other hand, anxiety may be because of the fear of death and dying process itself, existential crisis, a chronic coping or personality style such as avoidant or dependent personality, medication side effects, under treated symptoms such as pain, dyspnea, sepsis, withdrawal states from sedatives or opiates, and delirium. (chochinov, 2000). Anxiety can also occur in patients who are terminally ill as an adjustment disorder with anxious mood alone or in combination with depressed mood (massie, 1989). Adjustment disorder with anxiety is related to adjusting to the existential crisis and the uncertainty of the prognosis and the future (holland, 1989). Anxiety related to medical causes is frequently a consequence of poorly controlled pain. The patient who is in pain appears restless, tense, and may be agitated. Once the pain is controlled, the secondary anxiety subsides. A sudden onset of anxiety with respiratory distress or chest pain may signal pulmonary embolism or an acute cardiac event.

Among the drugs used in terminal stages, corticosteroids occasionally cause anxiety. Antiemetics such as metoclopramide and prochlorperazine, often cause profound akathisia and anxiety. Bronchodilators used for dyspnea can produce tremulousness and anxiety. Withdrawal states from opioid analgesics, benzodiazepines, alcohol, and barbiturates produce anxiety and agitation. Use of short acting benzodiazepines, such as lorazepam and alprazolam, may be associated with rebound anxiety.

Clinical features

Patients with anxiety may complain of tension or restlessness or exhibit jitteriness, autonomic hyperactivity, vigilance, insomnia, distractibility, shortness of breath, numbness, apprehension, worry or rumination. The somatic manifestations of anxiety may often be the presenting symptoms, and should be used as a cue to assess the psychological state of the patient. The patient may complain of many fears, like the fear of disease, fear of pain, fear of disability, fear of isolation, fear of loss of autonomy, and fear of death. In addition to these fears, there is also the retrospective anxiety aroused by the 'irreparable', compounded by feelings of guilt, regret, and despair with respect to lost opportunities, unfulfilled wishes, and missed areas of personal experience.

Management of anxiety disorders in terminally ill

The goals of palliative care cannot be achieved when the patient is overtly anxious. Thus management of anxiety is one important focus in end of life care. The management of significant anxiety in dying patients requires exploration of the possible source of anxiety and appropriate management according to the cause, which may include pharmacological intervention given along with psychological support or behavioral interventions. Treatment for anxiety in patients with advanced disease should be designed to provide rapid relief without placing excessive demands on the patient.

Pharmacotherapy

As a first step, a careful review of the patient's medical condition for anxiogenic medical disorders (e.g. Hyperthyroidism) or drug side effects should be conducted before starting any psychotropics. The management of anxiety symptoms begins with the clinician providing emotional support and adequate information to the patient. Pharmacotherapy primarily involves use of benzodiazepines, neuroleptics, antidepressants, and opioid analgesics.

Short-acting benzodiazepines, such as lorazepam and alprazolam, are used most often. Lorazepam is metabolized by conjugation in the liver and therefore, is safer to use in patients with hepatic disease. Alprazolam is metabolized through oxidative mechanisms in the liver and can exacerbate liver damage. Clonazepam, a longer acting benzodiazepine, is used in patients who experience breakthrough anxiety on short-acting drugs, and also in patients with anxiety who have seizures. Diazepam can be administered rectally to control anxiety and restlessness when oral administration is not possible (twycross & lack, 1984). The benzodiazepines are started at low dose, alprazolam (0.25-0.5 mg three times a day), lorazepam (0.5-2 mg three times a day), or diazepam (5-10 mg once a day).

Neuroleptics such as haloperidol or thioridazine, given in low doses, are used when benzodiazepines are contraindicated or do not control anxiety.

Tricyclic antidepressants are effective in treating anxiety with accompanying depression and panic attacks, but have limited usefulness in patients who are dying because of its delayed onset of action and anticholinergic effects; however, the sedative effects of these medications are useful (chaturvedi et. Al., 1994)

Other anxiolytics such as buspirone, a nonbenzodiazepine anxiolytic, and hydroxyzine have been tried, especially in respiratory distress associated with anxiety, as it lacks sedative and cns depressant effects. Antihistaminics and beta-blockers have also been tried when other drugs cannot be tolerated.

Psychosocial interventions

For providing psychological support, supportive, crisis-oriented psychotherapy is the best model. The physician must be able to discuss the meaning of information about the medical condition and treatment plan, listen to the patient's fears and concerns, and support adaptive ways of coping. Cognitive behavioral techniques seek to reduce anxiety by providing information and by teaching self-monitoring techniques, distraction, and relaxation. Cognitive approaches focus on thought processes and perceptions, while behavioral approaches focus on modifying behavior. The behavioral techniques most often use elements of muscular relaxation and cognitive distraction, passive breathing followed by passive or active muscle relaxation, and pleasant imagery.

Management of mixed anxiety and depression

Pharmacotherapy

For patients diagnosed to be having mixed anxiety and depression state, benzodiazepines, especially alprazolam, have been found to be very useful. Alprazolam is fairly well tolerated and has no anticholinergic side effects, however, some patients, especially the elderly may have sedation and behavioral disinhibition. Other classes of drugs like ssris, tcas can also be used in states of mixed anxiety and depression.

Issues about caregivers

The stress and psychological issues in the caregivers of terminally ill patients be they the doctors, nursing staff or family members are also important. Working with terminally ill patients produces a spectrum of painful emotions in the caregivers also. The caregivers can experience a whole range of negative emotions like helplessness, fear, guilt, anger, inadequacy, intolerance, vulnerability, over attachment, lack of control, ambivalence and frustration (barton, 1977). The nursing staff, doctors, family members, all are prone to stress and consequently may burnout while caring for the terminally ill patient. Besides the issues related to the patient per se, it is very important to help the caregivers in coping positively with the situation, either by counseling, medications, or relaxation techniques.

Conclusion

An anonymous 16th century author described the role of the physician as being 'to cure sometimes, to relieve often, and comfort always'. Despite technological advances, the cure oriented focus has not achieved its goal, and medicine is once again coming to recognize its part in addressing the concerns of the dying patient. Once the prospect of cure becomes an unreasonable expectation, the focus properly shifts to relief and reduction of suffering. With the acknowledgement of futility, the optimal care of the patient shifts from a focus of cure and care to a palliative one of care only. Within the domains of relief and comfort, psychiatry has an important role for improving the quality of life of terminally ill patients. The social ethics of medicine requires shared goals that allow patients a safe passage to a compassionate end. Though, we all die alone, our dependency on one another, especially during the dying process, makes each death intensely shared. It is the compassionate and competent manner in which care is delivered by physicians at end of life that helps the sickest of human beings to transcend suffering.

Anxiety and Depression in Pain Disorders

John Vijaysagar Kommu
Jacob K John

Case of chronic headache

Mr. Suresh, an 18 year old single male from a lower middle class Hindu nuclear family in West Bengal, presented to the outpatient department with headache of 9 years duration. The headache used to be moderate to severe in intensity, localised to the vertex region and continuous in nature. This headache was not associated with photophobia, perceptual abnormalities, blurring of vision or vomiting. The patient used to miss school often due to the disease. There was a gradual decline in his academic performance and he discontinued his studies after the 9th standard.

Over the years, the patient had been to several doctors including specialists like neurologists and ophthalmologists, and had undergone a battery of tests and investigations. Although several doctors had reassured him with negative investigation results, Suresh was not comforted and felt that the cause for his headache was undiagnosable. As there had been no improvement in the pain, he started expressing pessimism about his health and future.

In view of the above clinical picture a psychiatric diagnosis of Somatoform Pain Disorder was made. The salient features for diagnosis were

- A chief symptom of severe and distressing pain of prolonged duration (more than 6 months)
- Absence of underlying physical illness that can explain the nature and severity of pain.
- Persistent requests for medical investigations, in spite of repeated negative findings and reassurances by doctors.
- Some degree of impairment of social and family functioning.

Patients presenting with similar profiles are not uncommon in a doctor's clinic. Psychogenic pain can present with symptoms in any part of the body and can lead to a significant loss of function and resources. It is important to recognise this early and to start suitable and effective treatment to minimise the distress and expense. Often the primary physician can deal with **this** quite appropriately and cost effectively.

Pain is the commonest symptom in medical settings. It is usually associated with a pathological physical state. The International Association for the Study of Pain (IASP) defines pain as 'an unpleasant sensory and emotional experience associated with actual or potential tissue damage, or described in terms of such damage'. The purpose of this definition is to allow for the acknowledgment of pain whether an immediate physical cause was recognised or not. If patients report experiencing of pain, then they have pain, whether a physical lesion has been demonstrated or not. Subjectively, pain is a unitary phenomenon, but different etiological factors contribute to its cause and the physician is obliged to determine the importance of different etiological factors.

Epidemiology

Pain syndromes are among the most common problems presenting to general medical providers. Population surveys indicate that over 25% of community residents suffer from recurrent or persistent pain symptoms and that 2-3% experience fairly disabling pain syndromes. The recent WHO primary care survey found that approximately 20% of primary care patients suffered from persistent pain. Pain syndromes are approximately twice more common in women than in men.

In their study of outpatients in a psychiatry pain clinic, Anooshian et al., (1999) reported that 79% fit the diagnosis of pain disorder associated with both psychological factors and a general medical condition and 9% fit the diagnosis of pain disorder associated with psychological factors. In a study of

general medical and surgical inpatients evaluated by a psychiatric consultation-liaison service, King (1998) found that 51% of patients who reported having pain at the time of initial evaluation, fulfilled the diagnostic criteria for pain disorder.

Clinical features

In the ICD diagnostic system, somatoform pain disorder is defined as persistent pain without clear medical explanation. The DSM system specifies that 'psychological factors be judged to have an important role in the onset, severity, exacerbation and maintenance of the pain'. Both definitions are somewhat problematic. Recent findings regarding neural changes associated with a persistent pain raise doubts about the distinction between pain with and without a medical explanation. Recent research has attempted to avoid questions of unitary etiology and has examined the prevalence and correlates of persistent pain.

Pain symptoms are strongly associated with anxiety and depressive disorders. This relationship has been consistently demonstrated in both community and primary care studies across a broad range of cultural and socioeconomic divides. Psychological distress is most strongly associated with pain occurring at multiple sites and pain associated with functional impairment. Though, studies strongly support an association between pain complaints and psychological distress, it does not necessarily imply that pain is a consequence of psychological distress. Some studies find that the presence of psychological distress predicts the onset of pain syndromes; while others support the opposite relationship that persistent pain predicts subsequent psychological disorder.

Differential diagnosis of pain disorder

1. **Physical disorders**

   Many painful disorders have a well recognised organic pathology that accounts for the occurrence of pain (e.g. angina, carcinoma pancreas), but psychosocial processes tend to modify the severity of pain and associated disability.

2. **Mental disorders**

   - Mood and anxiety-related disorders like major depression and generalized anxiety disorder are by far the most common mental disorders associated with pain in most settings.
   - Complaints of pain occur commonly in each of the somatoform disorders and may be the predominant symptom.
   - Multiple physical complaints, often including pains at different sites, fluctuating from time to time usually for many years, is a characteristic feature of somatisation disorder.
   - In hypochondriasis, pain is a common complaint, and forms the focus of concern and overvalued belief about an unidentified disease.
   - Patients with any psychotic disorder may complain of pain, sometimes with bizarre descriptions of quality and delusional attribution. In overall terms these are less common.

3. **Pain syndromes of unknown origin**

In these syndromes, the primary complaint is that of pain. Psychosocial factors may contribute to predisposition, precipitation or course of these disorders. These are:

- Fibromyalgia (generalised pain).
- Tension headache (relatively localised pain).
- Temporo-mandibular joint dysfunction.
- Atypical facial pain.
- Atypical (non-cardiac) chest pain.
- Abdominal pain of psychological origin.
- Non-ulcer dyspepsia.
- Irritable bowel syndrome.
- Proctalgia fugax (ano-rectal pain).

Comorbidity

In pain clinic settings, the prevalence of mental disorders varies according to referral patterns, but about 30-40% of patients have depressive disorders. This is similar in those with and those without a relevant physical disorder.

Management

The specific aims of treatment vary with the individual and should be specified before treatment begins. There are four general aims

- To reduce stress
- To reduce disability
- To reduce symptoms and
- To limit inappropriate use of medical intervention

In the assessment of a patient with pain disorder, it is obvious that organic conditions have to be ruled out. A good rapport and doctor-patient relationship is of utmost importance. The physician or specialist should do a thorough physical examination and appropriate referrals and investigations.

If the clinical evaluation and investigations fail to reveal underlying physical illness, the same has to be conveyed to the patient in unambiguous terms. It should be clearly conveyed that getting consultations from multiple specialists ('Doctor- shopping') and repeating investigations is of no use. The treating physician has to acknowledge the patient's symptom and distress as real and should take care not to label the patient as a malingerer or psychologically weak. The physician should avoid prescribing analgesics for prolonged periods. It is also wise to avoid polypharmacy .The patient may have more than one problem and the physician has to be aware of this issue e.g. a patient with migraine headache may have a comorbid depression. While appropriate medication is not withheld, over-emphasis on medication may take away from other treatment initiatives.

It is unwise to assume that onset of pain in a patient with a previous psychiatric diagnosis is always psychogenic in nature. If a physician decides to refer the patient to a psychiatrist, it is important to have a good liaison with the psychiatrist to whom the patient is referred. The primary physician should also spend time and explain to the patient, as a significant proportion of patients are reluctant to consult a psychiatrist even when distressed with pain.

Management of pain is a multi-pronged, multi disciplinary process, where the skill of actively intervening is balanced by the skill of supporting and reassuring.

Several randomized trials have shown efficacy in pain management with treatment strategies, which typically include support and validation of pain, relaxation training, activity scheduling, reinforcement of nonpain behaviours and cognitive restructuring.

If the patient believes that the doctor acknowledges and cares about the pain and suffering, it is more likely to take the therapeutic alliance forward. Similarly simple measures like restructuring the day including more leisure and pleasurable activities and relaxation can alleviate anxiety and distress. The patient's key relatives can also be helped to encourage and reinforce non-illness and non-drug related behavioural responses to the pain. When successful, helping the patient to see these changes as helping in their lives would allow cognitive restructuring.

Antidepressants, particularly amitriptyline, have been found to a useful adjunct in pain management though benefits may not be uniform across pain conditions.

Conclusions

To summarize it has to be noted that careful assessment of pain and avoiding multiple referrals and investigations in a good therapeutic milieu will go a long way in alleviating the distress of the patient and also in minimising the enormous cost incurred in the management of the patient with pain disorder.

Anxiety and Depression associated with Major Surgery

Purvi Parikh
Santvana Sharma
Himani Ghoge
Shamsah Sonawalla
Rajesh Parikh

Case of below knee amputation

Mr. Xavier, a right handed, married, male patient, is admitted into the surgery ward for a below the knee amputation of the right limb due to gangrene of the right foot. Hospital staff and the patient's family notice that over a period of two weeks since hospital admission, the patient appears withdrawn, frequently expresses feelings of hopelessness, appears tearful, refuses to eat, and finds it difficult to sleep at night. Also, he refuses surgery and pleads with the doctors to let him die. The staff refer the patient to a psychiatrist and psychotherapist for evaluation and treatment. The patient receives a diagnosis of Major Depressive Disorder and is treated with antidepressants and individual psychotherapy. A week later, the patient notes feeling less depressed and accepts the need for surgery.

This case demonstrates that a surgical condition, such as an impending amputation, could act as a stressor, triggering a Major Depressive Episode. The psychiatric disorder, in this case depression, requires to be treated in addition to the surgical condition. In the above example, psychiatric symptoms precede a surgical event. However, they could occur as post-surgical sequelae, either immediately or delayed.

Mr. Xavier's diagnosis is supported by the following:

1. Symptoms (withdrawal from others, tearfulness, sadness, loss of appetite, sleep disturbances, suicidal ideation) are of a marked frequency, duration, and intensity and cause him significant distress.

2. Symptoms do not have an organic cause, and are not substance or medication induced, as ruled out by a physical examination, laboratory tests, and psychological assessment.

3. Symptoms are not merely accounted for by a grief reaction to amputation, or an adjustment disorder with depressed mood, and are not accounted for by any other psychiatric disorders.

When anxiety and/or depression occur in the context of major surgery, the foremost challenge is to ascertain whether the psychiatric symptoms and the surgical condition are related and how they might affect one another. Psychiatric symptoms might be related to a surgical condition in the following ways:

1. Surgical condition is the direct cause of anxiety/depression, for example, Coronary Heart Disease which sometimes needs by-pass surgery, is an independent risk factor for developing depression and anxiety (Lamberg, 2002).

2. A surgical condition exacerbates or precipitates a pre-existing psychiatric condition with no known physiological link, for instance, women undergoing hysterectomy develop exacerbation of pre-existing depression and anxiety in the absence of an underlying etiological factor (DSM IV, 1994).

3. Medications for a surgical condition cause symptoms of anxiety and depression as side effects, for example, anxiety and depressive symptoms in post-transplant patients due to the adverse effects of steroids.

4. Anxiety and depressive symptoms as a reaction to the stress and pain of a surgical condition, for example, depression, anxiety, and adjustment disorder in prospective amputees in response to the stress and pain of their illness.

The relationship between psychiatric symptoms and surgical conditions is bi-directional. For instance, in the preceding case example, the patient developed depression in response to the impending amputation. His depression, if untreated, might produce physical manifestations, such as aches and pains, which could complicate the surgical condition and may make the patient non-compliant for surgical treatment. An understanding of the relationship between psychiatric symptoms and surgery is likely to aid the treatment of both.

Epidemiology

Anxiety

The prevalence of Anxiety Disorders related to medical/surgical conditions vary for each specific condition. A syndrome similar to panic disorder is the most common clinical picture and a syndrome similar to phobia is the least common (Kaplan and Sadock, 1998). 83% of people with cardiomyopathy awaiting cardiac transplantation have symptoms of panic disorder, and about 25% of patients with Parkinson's disease and chronic obstructive pulmonary disease have symptoms of panic disorder (Kaplan and Sadock, 1998). The highest prevalence of symptoms of Generalized Anxiety Disorder in a medical condition seem to be in Grave's Disease. Reports have associated the development of Obsessive-Compulsive disorder with Sydenham's chorea and Multiple Sclerosis (Kaplan and Sadock, 1998). Psychiatric symptoms due to associated medical conditions might also affect the surgical condition. There is a 17% prevalence rate of symptoms of social phobia in patients with Parkinson's Disease (Kaplan and Sadock, 1998).

Depression

About 25%-40% of individuals with certain neurological conditions are likely to develop marked depressive disturbance at some point in their illness (Kaplan and Sadock, 1998). For general surgical/medical conditions, rates are far more variable, ranging from 60% in Cushing's syndrome to less than 8% in end-stage renal disease (Kaplan and Sadock, 1998). A study found that 15.2% of patients presenting for bypass surgery had elevated pre-operational levels of depression (Baker et al., 2001). It is found that 16-23% of the 12.5 million Americans with coronary artery disease also suffer from depression (Lamberg, 2002). Out of 504 cancer patients, 17.5% and 7.1% had pre-operative anxiety and depression respectively, and out of 226 patients presenting for surgery for epilepsy, 1/3 developed psychiatric symptoms before, during, and after surgery (Pascoe et al., 2000). Depression is common among patients recovering from multiple infarct bypass surgery where 1 out of 6 experience major depression before surgery and twice as many after surgery (Zeigelstein, 2001).

Etiology

Factors contributing to the development of anxiety and depression differ for each stage of the surgical condition. The stages are divided into: Pre-surgical, immediate post-surgical, and late post-surgical. Contributory factors may act individually or interactively to precipitate anxiety and depression in

patients undergoing surgery.

Presurgical stage

Contributory factors for Anxiety

Medical

Angina pectoris, hypertension, myocardial infarction, asthma, chronic obstructive pulmonary disease, pneumonia, seizure disorders, vertigo, anemias, hypothyroidism, menopause etc.

Drug-related

Hypotensive agents, bronchodilators (sympathomimetics), metoclopramide, withdrawal syndromes (alcohol, sedative- hypnotics), etc.

Psychiatric Disorders

- Generalized Anxiety Disorder
- Phobias
  - Blood-Injection-Injury Type
    - Situational Type.
    - Others knives, needles, enclosed spaces.
- Anxiety secondary to Pain Disorder.

Psychosocial factors

Anxiety as a personality trait, anxiety due to risk of surgery and complications, lack of social support, lack of education about surgery/surgical condition and surgery as threat to psychological equilibrium.

Contributory factors for Depression

Medical

Parkinson's disease, chronic subdural hematoma, temporal lobe epilepsy, stroke, hyperthyroidism, hypothyroidism, systemic lupus eythematosus, rheumatoid arthritis, uremia, hyponatremia, hypokalemia , viral and infectious hepatitis and AIDS.

Drug-related

Alcohol, cocaine, opiates, reserpine, propranolol, cimetidine, steroids, cardiac drugs, analgesics, antibacterials, antihypertensives, sedatives, hypnotics, antipsychotics, antiepileptics, and antiparkisonian medications (Bernstein et al., 2001).

Psychiatric disorders

- Substance abuse/ Dependence.
- Depression secondary to anxiety disorders, personality disorders, dementia, somatoform disorders etc.

Psychosocial Factors

- Life Events and Environmental Stress.
- Family Functioning.
- Pre-morbid Personality Factors.
- Grief and loss issues related to surgical condition (amputation, mastectomy, vasectomy, hysterectomy, etc).
- Sense of Hopelessness and Helplessness.
- Sense of Loss of Control leading to guilt and shame.

Immediate post-surgical

1. Anxiety and Depression may manifest immediately post-surgery due to side effects of drugs used during surgery e.g. sympathomimetic agents, vasopressor agents (anxiety inducing drugs), cardiac and antihypertensive drugs, sedatives and hypnotics, steroids and hormones (depression inducing drugs) etc. Other factors which contribute to anxiety and depression include stress and pain associated with surgery.

2. Anxiety and depressive symptoms associated with Post-operative delirium, hypoxia, and 'ICCU Syndrome'.

   Anxiety and depressive symptoms might occur in states of post-operative delirium. Patients with delirium often have abnormalities in the regulation of mood characterized by symptoms of unwarranted fear, apathy, and depression. Patterns of abnormal arousal in patients with delirium may also cause hyperactivity or hypoactivity resulting in symptoms of anxiety or depression respectively. Approximately 30% of patients in surgical intensive care units and cardiac intensive care units experience delirium (Kaplan & Sadock, 1998). Post-surgical patients might also suffer from anxiety and depressive symptoms due to hypoxia and sensory deprivation, a phenomenon known as 'ICCU Syndrome'.

3. Other post surgical factors that might cause anxiety and depressive symptoms include acute stress of surgery, post-operative pain, insomnia, pain medication, bodily loss of function, and infection.

Late post-surgical stage

Contributory factors for anxiety and depression

Medical / Surgical

- Surgical Complications.
- Use of Assisted Devices.
- Pace of Physical Rehabilitation.
- Side effects of Medications.
- Disfigurement.
- Amputation.

Psychiatric factors

- Sexuality - Fear of loss of sexual attractiveness, sexual dysfunction may cause anxiety and depression in surgeries such as hysterectomy, vasectomy, mastectomy, prostrate, and urethral surgeries.
- Altered body image.
- Stress of physical pain.

Psychosocial Factors

- Quality of Caretaker Support - Caretakers might behave with criticism, anger, hostility or over-involvement that could cause emotional distress for a patient.
- Change of Lifestyle.
- Family Functioning and Expressed Emotion - Pathological family behavior might significantly increase the emotional stress with which a surgical patient must cope.

Clinical features

The symptoms of anxiety and depression that are most likely to be associated with major surgery are as outlined below.

Anxiety

Features of anxiety vary with the subtype of the anxiety disorder. Symptoms of muscle tension, trembling, twitching, shakiness, muscle aches and sores might indicate a Generalized Anxiety Disorder. Somatic (shortness of breath, palpitations, gastrointestinal symptoms, autonomic hyperactivity) and depressive symptoms are also common manifestations of Generalized Anxiety Disorder (Kaplan & Sadock, 1998).

In individuals with Panic Disorder, panic attacks may follow excitement, physical exertion, or moderate emotional trauma like that of major surgery. The physical signs of a panic attack often resemble those of a medical condition like a 'heart attack' and include tachycardia, palpitations, dyspnea, and sweating. Individuals with panic disorder may become excessively apprehensive about the outcome of routine activities particularly those related to health issues. The experience of anticipatory anxiety between panic attacks is common. About 60% of patients with panic attacks also experience depression (Kaplan & Sadock, 1998).
Individuals with Phobias related to surgical conditions and settings are particularly vulnerable for the development of anxiety and depression. For instance, the blood-injection-injury type of phobia is cued on seeing blood or an injury or by receiving an injection or any other invasive medical procedure (DSM IV, 1994). A vasovagal attack is characteristic of this type of phobia and is often associated with fainting. Approximately 75% of such individuals report a history of fainting in these situations. Certain surgical conditions may be exacerbated as a consequence of phobic avoidance (DSM IV, 1994).

Individuals with Obsessive-Compulsive Disorder frequently avoid situations that involve the content of their obsessions, such as dirt or contamination. For instance, a person with obsessions about contamination may avoid hospitals or blood transfusions. Patients with Obsessive-Compulsive

Disorder may also express hypochondriacal concerns and they seek constant reassurance from physicians.

Depression

Common symptoms of depression include tearfulness, irritability, brooding, and anxiety. Complaints of physical aches and pains (headaches or joint, abdominal, or other pains) might be exaggerated in a surgical setting. Changes in food intake and rest associated with depression can aggravate co-existing medical illnesses, such as diabetes, hypertension, chronic obstructive lung disease, and heart disease and thereby complicate recovery from the surgical condition (DSM IV, 1994). About 97% of patients undergoing surgical procedures, complain of low energy, and 80% of patients complain of insomnia (DSM IV, 1994). In 90% of all depressed patients anxiety is a common symptom (DSM IV, 1994).

Depression is a risk factor for suicide and 15% of hospitalized, severely depressed patients die by suicide (DSM IV, 1994). Management of the general surgical condition is more complex and the outcome less favorable if there is associated and untreated depression.

Risk factors for pre and post surgical anxiety and depression

According to several research studies, risk factors associated with pre and post-surgical psychiatric symptoms include: pre-existing psychiatric disorders, age (younger patients are at greater risk), gender (women are at greater risk), and stressful social situations (Pascoe et al., 2000). Similar results are also indicated in a study assessing incidence and patterns of anxiety and depression in post-operative bypass surgery patients, that younger subjects experienced greater anxiety, and women have higher state and trait anxiety and nonsignificant levels of depression (McCrone et al., 2001).

An Australian study found that in pre-surgery cancer patients, the risk factors for anxiety and depression included amount of resting activity level, advanced stage of disease and being female (Pascoe et al., 2000).

Research in Iceland indicates that the increased length of waiting for Coronary Bypass Graft Surgery is associated with negative effects on work life and health status, and produced symptoms of shortness of breath, anxiety, depression, chest pain, uncertainty and stress which negatively affected outcome (Jonsdottir and Baldursdottir, 1998).

The pre-operational psychological state may be a risk factor for post-operational as well as late post-surgical psychological states. This is especially true for about one year into recovery (Boudrez and De Backer, 2001). Although there is no single variable that predicts late psychological state, a constellation of pre-operationally measured psychological variables such as somatic complaints, hostility (dysphoria), and anxiety are implicated (Boudrez and De Backer, 2001). Also, patients with coronary artery disease who have poor quantity and quality of sleep, and increased psychological symptoms before surgery, are related to increased symptoms after surgery (Gustaffsson-Edell, 1999).

About 20-25% of patients with associated medical conditions such as diabetes, myocardial

infarction, carcinomas, and stroke, might be at an increased risk for developing depression (Kaplan & Sadock, 1998). The risk factor is likely to be enhanced in surgical patients with associated medical conditions.

In summary, there are multiple risk factors ranging from gender to associated medical conditions that might increase the risk of developing anxiety and depression in surgical patients with pre-existing vulnerability.

Factors affecting post-surgical recovery

Psychiatric morbidity and associated psychosocial stressors may impede post-surgical recovery. A study on the effect of emotional response to surgery on functional recovery found that short-term recovery is predicted by surgical trauma, but long-term recovery is predicted by pre-existing psychological and physical functioning, and emotional response to surgery. The study underscores the importance of treating pre-operational anxiety independently as it could become a risk factor for increased bleeding during surgery and also lengthen post-operative recovery time (Caumo et al., 2001).

A study found that the psychosocial risk factors impacting recovery from spine surgery were pain sensitivity, depression, anger, and anxiety as indicated by their respective elevated scores on the Minnesota Multiphasic Personality Inventory (MMPI) (Epker and Block, 2001). After cardiac transplant surgery, symptoms of transient confusion, guilt, adjustment disorder, depression, and anxiety, are likely to slow recovery or undermine treatment compliance (Fitzsimons et al., 2000). For patients who have undergone Coronary Artery Grafting, anxiety, chest pain, and uncertainty negatively affect the outcome of treatment and these symptoms may be significant predictors of mortality over the next 25 months (Baker et al., 2001). Also, major depression is an independent risk factor for mortality in recovering surgical patients, especially those with associated medical conditions (Ziegelstein, 2001).

In a study of 712 adults presenting for elective surgery, neural blockade, or both, it was found that risk factors associated with poor post surgical functioning were: low physical function rating (ASA 111 status), history of smoking, moderate to intense post operative pain, high score on pain rating index, minor psychiatric disorder, pre-operational state anxiety, and negative future perception (Salmon et al., 2001).

Another study examined sleep, psychological symptoms, and quality of life in patients before and after surgery and their effects on functional capacity post-surgery. It found that patients with increased anxiety prone reactivity during 6 months following surgery had significantly more sleep disturbances, reduced energy and functional capacity, depression and/or cognitive/behavioral fatigue, and poorer quality of life compared with those without such reactivity (Gustafsson-Edell, 1999).

The above research highlights the bi-directional relationship between psychiatric symptoms and surgical conditions, and emphasizes the importance of pre-surgical psychiatric screening.

Management

Assessment

The first step towards effective management of depression and anxiety is their assessment vis-à-vis the surgical condition. It is important to distinguish whether anxiety and depressive symptoms are independent of, a reaction to, or caused, exacerbated, or precipitated by the surgical condition. It is also important to assess whether psychiatric symptoms are manifestations of surgical/medical problems. For instance, some illnesses such as Acquired Immune Deficiency Syndrome (AIDS) and pancreatic cancer often present with only depressive symptoms initially.

The major goals of psychiatric evaluation are recognition of the psychological determinants of behavior and symptom classification. Its main components include psychiatric history, mental status exam, psychological testing and physical assessment.

A careful and comprehensive assessment of multiple factors is necessary to make a judgement of the relationship between psychiatric symptoms and the surgical condition. Although there are no infallible guidelines for determining whether the relationship is etiological, the following chart presents several considerations for guidance.

To establish on the basis of a psychiatric history and mental status exam whether symptoms are indicative of a psychiatric disorder or of a general medical condition

Symptoms are indicative of a primary psychiatric disorder if there exists

1. A previous history of psychiatric illness.
2. Family history of psychiatric illness.
3. Symptoms in excess of those caused by a medical condition and result in significant distress.
4. Identifiable psychosocial stressor.

Symptoms are indicative of a general medical condition if there exists

1. History of organic antecedents like head injury, seizures, unconsciousness, substance use.
2. Neurological signs and symptoms.
3. Laboratory findings such as blood tests, MRI, SPECT scan, etc.
4. Temporal association between the onset, exacerbation or remission of the general medical condition and psychiatric symptoms.
5. Remission of psychiatric symptoms after removal of medical condition/ medication / substance use.
6. Presence of features atypical of a primary psychiatric disorder (atypical age of onset, atypical course).
7. Evidence from literature that suggests a direct association between a certain general surgical/medical conditions and psychiatric symptoms.

Pharmacotherapy

A combination of medical treatment and psychotherapy is the best therapeutic approach. The treatment approach is likely to depend on the relationship of anxiety and depression to the surgical condition as per the psychiatric assessment and associated findings. In general, the aims of treatment are:

1. To alleviate psychiatric symptoms.
2. To curtail the impact of psychiatric symptoms on surgical condition.
3. To prevent adverse effects of surgical condition on psychological health.

According to the APA (American Psychiatric Association), while the Quality of Life Index is usually high in post-surgical patients, psychological functioning usually lags behind physical functioning (Lamberg, 2002). The need for appropriate and complementary treatment services cannot be emphasized enough.

Considerations in administering psychotropic drugs to medically ill patients include a potentially increased sensitivity to a drug's side effects, impact of drug's side effects on the surgical condition, either increased or decreased metabolism and excretion of the drug, and interaction with other medications (Kaplan & Sadock, 1998). However, newer and safer medications with fewer adverse effects and relatively lower toxicity in overdose are now available for depression and anxiety. The newer medications include SSRIs (Serotonin-Specific Reuptake Inhibitors) such as Fluoxetine, Fluvoxamine, Paroxetine, and Sertraline, SNRIs (Serotonin Noradrenergenic Reuptake Inhibitors) such as Venlafaxine, and other drugs such as Mirtazepine, Bupropion, Trazodone, Nefazodone etc (Kaplan & Sadock, 1998).

Judicious use of these medications, in light of their risk-benefit ratio and careful monitoring can be useful in treating individuals with co-existing psychiatric and surgical conditions.

Psychosocial intervention

Treatment intervention may occur at any surgical stage (pre, post, late) and should follow an affirmative psychiatric evaluation. Intervention can be prophylactic, short-term crisis oriented, and long-term rehabilitative.
For patients with anxiety and depression at the pre-surgical stage, intervention may include information sharing and education about what to expect from surgery and emphasizing the importance of maintaining a positive attitude towards the outcome of surgery.

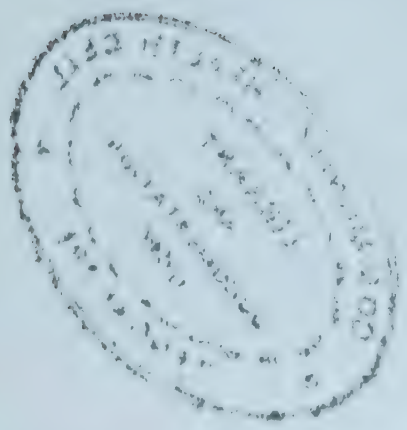
Immediately after surgery, patients who are anxious and depressed benefit from direct communication with staff, reassurance, discussion of feelings, empowerment and a symbolic sense of progress.

Long-term psychological functioning depends on mature life adjustment, an increased capacity to adjust to the presence of the disease, decreased use of secondary gain associations with the illness, and an increased capacity for physical and occupational functioning. Long-term rehabilitation should include caretaker support since they assume a heavy burden once the patient leaves specialist care. Investment in caretaker support could facilitate patient recovery and rehabilitation.

Common therapeutic approaches range from insight oriented therapies (Existential therapy, Rogerian therapy) to Behavioural therapies (Cognitive Behavioral Therapy, Biofeedback, Relaxation). The modes of therapy include individual, family, inpatient or outpatient group therapy, and support networks.

Conclusions

A unanimous conclusion among experts based on research studies is the importance of including a pre-surgical psychiatric screening in the medical diagnostic procedure. It is necessary for physicians to anticipate, recognize, and address psychiatric symptoms for any surgical treatment to be effective. Early recognition of psychiatric symptoms helps identify the cause, direct appropriate intervention, prevent surgical complications and improve post-surgical prognosis. Factors that minimize the likelihood of developing anxiety and depressive symptoms before and after surgery include informed consent, patient education about surgical outcomes, encouragement to maintain a positive attitude, adequate pain management combined with the use of preventive coping skills for pain and functional losses and constructive ongoing family support.



Anxiety and Depression associated with Sexual Dysfunctions

Henal Shah
Roop Majli

Case of Dhat Syndrome

Mr. Sinha, a 22 year old laborer from Bihar, presently working in Mumbai was referred to the Psychiatric Outpatient Department from the Medicine Department. His major complaints for over a year were tiredness, backache, bodyache and pains, listlessness and fatigue. He also had loss of appetite and difficulty in falling asleep. His physical ailments has been casting a shadow of anxiety causing despondency and reducing his enthusiasm for life. On further questioning, Mr. Sinha revealed that he was concerned about an excessive loss of semen due to night emissions and masturbation. He attributed his fatigue and weakness to this loss and also feared that loss of semen would cause his penis to shrink and lead to impotency. He was also concerned about his forthcoming marriage and feared that he would be unable to perform sexually and satisfy his wife.

Mr. Sinha was diagnosed to be suffering from Dhat Syndrome, a commonly seen culture bound syndrome. Dhat syndrome arises from the erroneous belief that semen is more precious than blood and 40 drops of blood are needed to form a single drop of semen. Thus, any loss of semen would result in loss of vital energy (Nakra, 1978). In this case, the treatment consisted of psychoeducation, where his erroneous belief system was corrected. His masturbatory anxiety and guilt were dealt with through counseling.

Epidemiology

Symptoms of anxiety and depression are pervasive among patients with sexual dysfunctions. Impotence or erectile dysfunction, premature ejaculation and dhat syndrome are among the most commonly seen sexual dysfunctions. Bagadia et al., (1972) studied patients with sexual complaints attending a psychiatric clinic. They found that 48% of these patients complained of impotence and 34% had premature ejaculation. It was noted that patients often presented with more than one type of sexual dysfunction and 75% of them scored high on scales measuring depression. 80-90% of patients diagnosed with dhat syndrome had symptoms of anxiety and depression. 50% of the interviewed men failed to consummate their marriage on the wedding night. Most of them attributed this to intense fear, anxiety and guilt.

35-40% of college going students and young adult males complain of premature ejaculation. Often this is due to anxiety about the sexual act and negative conditioning. This usually happens when they are with a commercial sex worker, where there is a fear of discovery and pressure to perform quickly.

Another group of patients who suffer from sexual dysfunctions are those who have anxiety or depressive disorders. In a study carried out by Montgomery (2001), it was reported that nearly 83% of depressed men and 53% of depressed women reported loss of libido. Thus anxiety and depression are bi-directionally linked with sexual dysfunctions.

Etiology

The interplay between sexuality and anxiety and depression can arise from the following scenarios

1. Sexual dysfunctions causing anxiety and depression.
2. Anxiety and depression causing sexual dysfunctions.
3. Drugs used for the treatment of anxiety and depression causing sexual dysfunctions.

Sexual Dysfunctions causing Anxiety and Depression

Sexual dysfunctions can be due to biological and psychological factors. Biological factors may be implicated in less than 5% of men with sexual dysfunctions, while in the remaining 95% the main causative factors are psychological in nature.

Anxiety and Depression often accompany sexual dysfunctions irrespective of the etiology. More often than not, the presenting features are anxiety, depression or somatic complaints. It is only on skillful questioning that these complaints are revealed and discussed.

Anxiety and Depression causing Sexual Dysfunctions

Excessive anticipatory anxiety about sexual performance itself can lead to sexual dysfunction. Similarly predominant symptoms of depression such as loss of enthusiasm and loss of interest in pleasurable activities lead to a decrease in desire to indulge in sexual activities. After the onset of depression, frequency of coitus as well as sexual satisfaction, decrease significantly. They may also experience occasional increase in premature ejaculation and erectile dysfunction.

Altered cognition causes low self-esteem and doubts about ones attractiveness. These symptoms aggravate the anxiety, damping sexuality and its pleasures.

Sexual dysfunctions associated with anxiety and depression improves as the cloud of anxiety and depression passes over.

Drugs used for the treatment of Anxiety and Depression causing Sexual Dysfunctions

The commonest class of antidepressant drugs used, are the Selective Serotonin Reuptake Inhibitors (SSRIs) such as fluoxetine, fluvoxamine sertraline, paroxetine and citalopram. More than 30% of patients on SSRIs complain of sexual side effects (Bezchlibnýk-Butler & Jeffries, 1999). Among SSRIs, sexual dysfunction seems to be more frequently associated with paroxetine and less frequently associated with fluvoxamine and sertraline. Failure to ejaculate in men and anorgasmia (inability to have an orgasm) in women are the most common complaints with SSRIs. The Tricyclic antidepressants also cause sexual side effects ranging from 2% with amitriptyline to nearly 30% with imipramine. The newer range of antidepressants, like venlafaxine also cause impairment in more than 30% of the user groups. Mirtazapine and bupropion have fewer sexual side effects. However, almost all antidepressants cause sexual side effects ranging from decrease in libido to ejaculatory disturbances, impotence and anorgasmia. Antipsychotic drugs, which sometimes may be given to this class of patients, may also cause sexual dysfunctions.

The important word of caution however is that before ascribing medication as the cause of sexual dysfunction one should consider the role of the disease and also assess premorbid sexual functioning.

Contributory factors for Anxiety and Depression associated with Sexual Dysfunctions

Many of the sexual complaints arise due to a combination of myths, ignorance and taboos. In our society sexuality is not an acceptable urge and the taboo's handed down exert a strong restraining hold. Religion also extols the virtue of celibacy. Traditional medicine also advises restrain. Sexuality as a means of pleasure and not procreation, is not accepted easily. This creates immense feelings of guilt in people. Ignorance is another blinker blinding and misleading many people. There is no formal and informal means of obtaining factual information. This leads to apprehension and spreading of myths.

Patients suffering from dhat syndrome believe that semen is vital, precious and of limited quantity. In fact 40 drops of blood go in making a single drop of semen. Loss of semen through night emissions, masturbation or excessive coitus is thus harmful. It would lead to impotency, physical malaise and penile shrinking.

Fear of passing 'dhat' after mictuiration or defecation causes apprehension. This results in anxiety and presentation of dhat syndrome with myriad somatic complaints. Commonly associated with dhat syndrome is fear of impotency. The high anxiety so aroused then further negatively affects the performance and the viscious cycle carries on. Masturbation due to its cultural and religious taboo and due to loss of semen is considered detrimental. Thus a healthy way of gratification, in the absence of a partner is avoided or when indulged in, causes immense anxiety, shame and guilt. It is falsely believed that sexual satisfaction correlates with the dimension of penis. Thus when one negatively views ones self, insecurity about ones ability to perform well, and satisfy the partner arises. Lack of privacy and conditioning of having hurried intercourse in embarrassing situations, pave the path for premature ejaculation and impotency.

Clinical features

Some of the common sexual dysfunctions encountered in clinical practice include, Erectile Dysfunction (impotence), Premature Ejaculation and Dhat Syndrome.

Erectile Dysfunction

The chief complaints in erectile dysfunction are a complete or partial failure to attain an erection or to maintain an adequate erection till the completion of sexual act. Impotence is the chief complaint in more than 50% of men being treated for sexual dysfunction. The incidence of impotence increases with age. Masters and Johnson have reported that most men over the age of 40 have a fear of impotence, which reflects the masculine fear of loss of virility with advancing age. In some men, this fear can lead to such extreme anxiety as to cause sexual dysfunction. In addition, episodes of impotence are reinforcing, with the men becoming increasingly anxious before each sexual encounter.

Premature Ejaculation

In premature ejaculation, men persistently or recurrently achieve orgasm and ejaculate before they wish to. There is no definite time frame to define premature ejaculation. The diagnosis is made when a man regularly ejaculates before or immediately after entering the vagina. Premature ejaculation is

more commonly reported among highly educated men vis-a- vis the less educated since they seem to be more concerned with partner's satisfaction. 35-40% of men being treated for sexual dysfunctions have premature ejaculation.

Dhat Syndrome

Dhat syndrome prevails primarily in the uneducated population and significant percentage of these patients present primarily with somatic complaints like aches and pains, weakness, perceived loss of strength and weight loss. Dhat syndrome and masturbatory guilt often co-exist since loss of semen through masturbation is believed to lead to loss of strength, vigor and disease. Popular misconception that masturbation is unnatural and immoral also leads to severe guilt and anxiety, which may lead to sexual dysfunction.

Often erroneous beliefs and misconceptions like the sexual act being only a means of procreation and deriving pleasure from it is sinful, can in the long-term cause negative conditioning. This coupled with fears of pregnancy and social stigma can lead to disorders like Dyspareunia (genital pain occurring before, during or after intercourse), Vaginismus (painful contraction of the lower portion of the vagina) and Frigidity in women.

Other causes like distress over homosexual tendencies and non-consummation of marriage can also precipitate sexual dysfunctions.

Management

Psychosocial intervention

Management of sexual dysfunctions and associated anxiety involves, sex education, behaviour therapy and sex therapy.

Sex education

It involves explaining normal anatomy, physiology and discussing myths that exist. It is usually in the form of an interactive group.

Behavior therapy

Behaviour therapy involves relaxation therapy, which helps to decrease high anxiety and desensitization techniques to counter the fear of impotency.

Sex therapy

Sex therapy involves sensate focus therapy for erectile dysfunction and specific techniques such as squeeze and stop start method for premature ejaculation.

The sensate focus technique is used to decrease anxiety especially about outcome and to learn to enjoy, and feel comfortable and in control, during coitus. It is a staged procedure with limits, which help in mastering pleasure without anxiety. The stages lead from non-genital to genital contact and finally to containment. During these stages just before impending orgasm, the partner squeezes the

tip of the penis. Once the urge to climax decreases the love making proceeds again. Alternately the male just prior to ejaculation is asked to stop for a while (about 30 seconds), gain control and start again. These methods counter premature ejaculation.

Pharmacotherapy

Anxiolytics and antidepressants may be required for the treatment of associated symptoms of anxiety and depression. Occasionally antidepressants have been used in the treatment of premature ejaculation as they delay the process. Clomipramine, fluoxetine and paroxetine are some of the most commonly used drugs, for this purpose.

Other methods such as use of intercavernosal injections of smooth muscle relaxants such as papaverine and phenoxybenzamine and hormones are being explored, especially for organic causes of erectile dysfunction.

Often it is a difficult task to treat psychosexual dysfunctions with counseling alone. The patients are dissatisfied with the explanation given during sex education. There are reports of patients having better compliance and outcome on addition of anxiolytics. Nakra et al., has recommended empathic listening, a non-confrontational approach, reassurance and correction of misbeliefs along with use of placebo, anxiolytics and antidepressant drugs whenever required.

In case of sexual dysfunctions being induced by medications, the dose can be reduced or the medication changed. Various other medications such as use of cyproheptadine (4-16 mg/day), yohimbine (5.4-16.2 mg/day) and bethanechol (100-400 mg/day) for impaired erection; neostigmine (7.5-15 mg/day) to improve ejaculatory function and buspirone (15-60 mg/day) can also be tried. The role of sildenafil (25-100 mg) is not yet clear though it has been used. Sildenafil does play a role in biologically induced ejaculatory disturbance and in iatrogenic dysfunctions. The role of sildenafil in purely psychogenic dysfunctions is not yet well established.

Insomnia and Use of Benzodiazepines

Vihang Vahia
Pallavi Pandit
Purvesh Madhani
Siddique Ahmed

Oh gentle sleep, Nature's soft nurse how have I frighted thee.
-Henry IV, William Shakespeare

Case of insomnia

Mr.Dinesh a 38 year old married executive complained of non-restorative sleep for 3 months. He complained of difficulty in initiating sleep and early morning awakening. He felt 'low' throughout the mornings 'dragging' himself to work. On probing, he revealed that he was worried about his own and his family's future and finances. A young MBA had been given a promotion, bypassing him. He now wished that he had done his MBA. He had stopped playing tennis and preferred sitting at home by himself since the onset of the current symptoms.

This is a prototype case of patients, visiting primary care physicians with presenting symptom of inadequate sleep. Although easily recognizable by psychiatrists as a case of major depression, it entails taking a detailed history to indubitably identify the same. Mr.D fulfills the ICD-10 diagnostic criteria for Depressive episode.

Insomnia

Numerous underlying pathologies may present with symptoms of insomnia. The treating physician is required then to disentangle this arachnoidal web to tackle the central sleep-depriving beast itself. Sleep is a reversible state of unresponsiveness and tranquility. Insomnia is the complaint of inadequate or non-restorative sleep. The difficulty could be of initiating and maintaining sleep or early morning awakening (APA, 1994).

Epidemiology

A large number of adults who complain of insomnia are found to be suffering from a psychiatric ailment. Insomnia often acts as a herald for psychiatric illness. About 30% of adults may complain of insomnia, 40% of whom may have a psychiatric ailment (Benca, 1996).

Clinical features

Persistent insomnia leads to many complications. Non-restorative nocturnal sleep leads to fatigue with resultant drop in work performance. It may also lead to anxiety, irritability and other more severe mood disturbances. Prolonged sleep disturbances may lead to cognitive disturbances such as reduced ability to concentrate and focus, impaired attention and forgetfulness. Seemingly innocuous, insomnia can extract a heavy price from us. Most people are very concerned about sleep. Insomnia even of a short duration leads to great concern, arousing anxiety which often fuels the insomnia leading to viscious cycle.

Evaluation of a patient with insomnia

Insomnia is a symptom. It is not a disorder in itself. In order to treat insomnia it is essential to diagnose the accompanying medical or psychiatric disorder. Clinical diagnosis is based on detailed evaluation of the insomnia.

The mandatory assessment of insomnia includes:

1. Sleep History

A general characterization of the insomnia i.e. its duration, severity, variation, daytime consequences and following specific questions

a) Do you have difficulty primarily in
   - Falling asleep
   - Staying asleep
   - Waking too early
   - Combination of the above
   - All of the above (pan insomnia)?
b) How long are you awake at each juncture?
c) When did your insomnia first start?
d) How many nights per week/month does your insomnia occur? Is it related to season, menstrual cycle or any other cyclical factors?
e) What are the daytime consequences of your sleep problems? (e.g. fatigue, irritability, concentration problems)
f) Is your occupation causing the sleep problems? (Work schedule, shift duty, jetlag)

2. Sleep diary

It may be of a special value in estimating the severity of the problem, the night- to-night variability and presence of maladaptive sleeping behavior such as any-time naps or spending excessive time in bed (more than 8 hours). Sleep diary also keeps track of compliance with behavioral intervention & response to treatment.

3. Sleep and Psychological Rating Scales

Epworth Sleepiness Scale rates the chance of dozing in the following situations (Johns, 1991)

- Sitting and reading.
- Watching television.
- Sitting inactively in a public place.
- As a passenger in a car for an hour without a break.
- Lying down to rest in the afternoon.
- Sitting and talking to someone.
- Sitting quietly after lunch.
- Without alcohol.
- At a traffic signal in a car.

0- No chance of dozing
1- Slight chance of dozing
2- Moderate chance of dozing
3- High chance of dozing

A score of more than 16, indicate daytime somnolence, which may be the cause or effect of insomnia at night.

4. Focussed physical examination

Certain sleep disturbing pathologies such as COPD, asthma or restless leg syndrome are assessed by thorough general examination.

5. Blood tests

Blood investigations may help to rule out subtle manifestations of thyroid disease, iron deficiency and Vitamin B12 deficiency (restless leg syndrome) (Mahowald, 1996).

6. Actigraphy and Static change beds

These methods measure body movements in a measured total sleep time.

7. Polysomnography

It is considered the gold standard for measuring sleep. EEG, EOG, EMG, ECG, pulse oximetry are used to reveal a variety of findings like periodic limb movement disorder, sleep apnea, narcolepsy etc., (Coleman et al., 1982).

Etiology and Management

I) Primary Insomnia

About 15% of cases of insomnia, present without an underlying cause. Hence the term primary or idiopathic insomnia. There is difficulty falling asleep & multiple awakenings. Often people are preoccupied with getting enough sleep.

Management

1. Pharmacotherapy

It includes cautious use of benzodiazepines not exceeding 2 weeks owing to the inherent risk of tolerance & withdrawal phenomena. It is pertinent to note that benzodiazepines produce NREM sleep and they suppress dreams thereby altering the texture of normal sleep. Prolonged use of benzodiazepine produces other psychiatric disorders attributed to REM suppression. Abruptly omitting benzodiazepines may produce rebound insomnia. With the exception of sudden insomnia attributable to traumatic life events, it is recommended that antidepressant drugs with sedative properties be preferred to benzodiazepines, as the sleep induced by the antidepressant drugs resembles the normal sleep. Non bezodiazepines like zolpidem and zopiclone may also be preferred.

2. Sleep Hygiene

This is the recommended non-drug strategy that ought to be implemented by a patient of nonrestorative sleep (Morin et al., 1994; Regestein, 1998).

- Arise at the same time daily.
- Limit daily in-bed time to the usual amount present before the sleep disturbances.
- Discontinue CNS-acting drugs (caffeine, nicotine, alcohol, and stimulants).
- Avoid day time naps (except when sleep chart shows they induce better night sleep).
- Establish physical fitness by means of a graded program of vigorous exercise early in the day.
- Avoid evening stimulation; substitute radio or relaxed reading for television.
- Try hot, 20-minute, body temperature-raising bath soaks near bedtime.
- Eat at regular times daily; avoid large meals near bedtime.
- Practice evening relaxation routines, such as progressive muscle relaxation or meditation.
- Maintain comfortable sleeping conditions.

II) **Insomnia related to Psychiatric Disorders** (Benca, 1996)

1) **Major depressive disorder:** There is difficulty in maintaining sleep. Frequent awakenings during the second half of the night and early morning awakening associated with worsening of depressive features characterize the insomnia seen in this disorder. Management includes antidepressants for remission of the underlying disorder. Neurovegetative symptoms viz. sleep and appetite, respond before improvement in mood (Neylan, 1995).

2) **Manic episode:** Patients in mania could present as short sleepers. They may have difficulty in falling asleep, but only few voice their complaint. Despite less time spent in sleep they awaken refreshed. Mood stabilizers, antipsychotics and short-term administration of clonazepam or lorazepam cause remission.

3) **Schizophrenia:** The sleep improves with antipsychotics used to treat psychosis. ECTs cause behavioral control and reduce the perceptual disturbances, which in turn may promote sleep.

4) **Panic disorder:** Sleep onset and intermediary sleep maintenance insomnia may occur. Paroxysmal awakenings due to sleep panic are common during the transition between stages two and three. SSRIs or TCAs with short term use of benzodiapines control the panic symptoms thereby restoring normal sleep.

5) **Other Anxiety disorders:** Generally patients present with sleep onset insomnia due to excessive anxiety & apprehensive expectations about one or more life circumstances. In OCD the indulgence in ritualistic behaviors hamper initiation of sleep. Nightmares in PTSD could cause frequent awakenings and Nonrestorative sleep. Management includes treating the underlying pathology.

6) **Substance related insomnia:**

a) Alcohol: Causes difficulty in initiating and maintaining sleep and produces abnormality in REM sleep architecture.
b) Benzodiazepines: Long-term use causes fragmented sleep, an increase in stages 1 and 2 NREM and decrease in stages 3 and 4 NREM sleep. Withdrawal is associated with initiation disturbances with rebound insomnia.
c) Nicotine: Long-term use interferes with sleep onset. Smokers typically sleep less than non-smokers.

d) CNS stimulants: Cause sleep-onset insomnia.

III) Insomnia related to Neuro-Psychiatric Disorders (Vitiello et al., 1992)

1) **Dementia:** Causes altered diurnal rhythm, which results in nocturnal insomnia and daytime somnolence. Sundown syndrome is characterized by nocturnal confusion.

2) **Parkinsonism:** Here sleep is reduced in the more rigid form and obstructive sleep apnoea may supervene due to autonomic disturbances that set in.

IV) Insomnia related to Medical Disorders

1) **Obstructive sleep apnoea syndrome:** Seen in elderly and obese people characterized by intermittent snoring and gasping. Management involves use of nasal continuous positive airway pressure.

2) **Sleep related GER:** Regurgitation of stomach contents into esophagus during sleep causes distress. Management includes head elevation and H2 antagonists (Hoffstein et al., 1992).

3) **Sleep related asthma:** It causes random coughing episodes leading to multiple awakenings.

4) **COPD:** Impairment of airflow through the lungs leads to heightened sleep latency and disturbed sleep (Cormick et al., 1986).

5) **Peptic ulcer disease**: Nocturnal epigastric pain interferes with sleep.

6) **Periodic limb movement disorder (Nocturnal myoclonus):** Repetitive contractions of leg muscles causing frequent awakenings and daytime somnolence. Benzodiazepines and levodopa may be useful.

7) **Restless legs syndrome:** Deep creeping sensations inside the calves cause an irresistible urge to move the legs. Leg movement & massage relieves the symptoms. Benzodiazepines, carbamazepine and levodopa may be tried.

8) **Narcolepsy:** It is characterized by tetrad of cataplexy, sleep paralysis, hallucinations and daytime sleep attacks. Treatment includes forced naps, psychostimulants to abort daytime somnolence and tricyclic antidepressants to reduce cataplexy. Modafinil improves psychomotor performance.

V) Insomnia related to environmental conditions

1) **Adjustment sleep disorder:** Transient insomnia occurs in relation to acute stress.

2) **Inadequate sleep hygiene disorder:** Some habits are self-defeating in promoting adequate sleep, e.g. excessive socializing at night, drinking, smoking.

3) **Circadian rhythm disorders:** Drastic changes in sleep-wake schedules are usually seen in shift workers and long distance travellers.

'The best cure for insomnia is to get lots of sleep!'
- W. C. Fields.

'Now that the gates of the day are closed, leave tomorrows problems for tomorrow'
-Max Ehrmann.

Use of Benzodiazepines

Indiscriminate or unsupervised benzodiazepines use cause a state that was described in a different context by H.R.George and I.W. Shanklin on November 16th 1900 (Lancet, 24/11/1900 pg. 1531) when they said 'thus the mode of cure make sure of the renewal of the disease: surely a sad irony on modern method'

Benzodiazepines were synthesized in the 1950's (Lader, 1991). Initial studies had indicated that they were superior to barbiturates on the measure of dependence and cognitive adverse effects. Over the five decades of their existence, they have proven their efficacy in their quick control of symptoms of anxiety and their sleep inducing properties. However, their long-term use (beyond four weeks) is associated with the following problems (Lader, 1994):

1. Dependence

Unlike barbiturates, benzodiazepine users do not escalate the dose. However, they become reluctant to taper or omit their doses. Hindmarch described this phenomenon as partial dependence.

2. Dose tapering

Post taper anxiety and insomnia are reported in patients who might have used this drug for a couple of months. This post taper syndrome mimics the initial symptoms, which had prompted the drug prescription. This rebound of symptoms contributes to continued usage of the drug.

Benzodiazepine discontinuation syndrome was described in the 1960's by Leo Hollister as 'an abrupt discontinuation of high doses of chlordiazepoxide or diazepam could lead to a discontinuation syndrome'. Therapeutic doses given for a long time may also lead to it.

The minor discontinuation syndrome consists of anxiety, insomnia and nightmares, while the major discontinuation syndrome is characterized by grand mal seizures (Petursson & Lader, 1984), psychosis, hyperpyrexia and death.

3. Deficits

Benzodiazepine use is associated with difficulty in new learning. A peculiar symptom of state dependant learning wherein learning that might have occurred under the effect of benzodiazepines can be recalled only under its influence (Hantouche et al., 1992).

4. Disinhibition

Once in a while, everyone gets an impulse to indulge in socially unacceptable acts. A proper orientation into reality and social inhibition creates an anxiety, which inhibits implementation of such an impulse. Alcohol induced disinhibition facilitating implementation of unacceptable impulses is a common experience. Similar disinhibition also occurs amongst benzodiazepine users. They do not experience the anxiety, which would either be associated on the implementation of unacceptable impulses. White-collar crimes are frequently concomitant with benzodiazepines.

The American Psychiatric Association constituted a task force on benzodiazepines (Salzman,1991). Their guidelines, though not unanimous, identified that two third of the adult who use benzodiazepines, take it for 60 days or less.

Benzodiazepine abusers or indiscrete users fall into four groups:

- Elderly medically ill patients receive the drug from non-psychiatrists.
- Patients of panic or agoraphobia who are prescribed benzodiazepines by psychiatrists seldom escalate the dose.
- Psychiatric and general medical patients with recurrent dysphoria may escalate their dose or exhibit concurrent alcohol use or abuse of other substances.
- In patients of chronic insomnia, the American Psychiatric Association Task Force mentions that despite absence of confirmed efficacy of benzodiazepines, if taken for more than 30 consecutive nights, some patients continue to use the drug for longer period. The Task Force suggests that the continued use may be to, prevent rebound insomnia.

Recommendations

The Royal College of Psychiatrists Committee on safety of medicines recommends limited use of benzodiazepines. Benzodiazepines can be used as antianxiety drugs in patients who have severe anxiety, anxiety associated with a severe impairment or anxiety and insomnia associated with a psychiatric or general medical condition. Benzodiazepines are almost always contraindicated in case of mild anxiety or mild insomnia when an anxiolytic or zopiclone/ zolpidem may be used. When starting a patient on benzodiazepines, the minimum effective dose should be used. Long-term use of benzodiazepines is not recommended due to tolerance and dependence. Once sleep has been reestablished, doses should be tapered gradually to prevent withdrawal effects. When used as a hypnotic, benzodiazepines may be used only intermittently.

Precautions

When prescribing benzodiazepines, the following precautions must be taken. Benzodiazepines must be avoided in suicidal patients (or used in combination) to prevent overdose. The medication if given must be tapered under supervision. Benzodiazepines are not recommended for patients of phobias, obsessions and chronic psychosis. Benzodiazepines may obstruct adaptation to the changed environment following bereavement. They must be prescribed judiciously. Disinhibition i.e., reduced control over one's thoughts, behaviours and feelings, accompanies benzodiazepine use, similar to the effects of alcohol. This benzodiazepine disinhibition may precipitate suicide in depressed patients. These patients may also exhibit aggressive outburst in a vulnerable patient, which is referred to as psychokinetic stimulation.

Benzodiazepine Toxicity

Benzodiazepines potentiate the inhibitory effect of GABA on the CNS. Toxicity may be evident within 30 minutes of an overdose. In the young and middle-aged adults, the toxicity is mild unless other sedatives or alcohol is ingested with benzodiazepines (Salzman, 1991). Ataxia, sedation and impairment in psychomotor speed may occur.

Amongst the elderly, cognitive impairment may be associated with both acute and chronic therapeutic doses of benzodiazepines. Predisposition to falls is also common. Occasionally paradoxical excitation may occur early in the course of overdose. Coma and respiratory depression supercede in severe cases. Constricted pupils and excretion of metabolites in the urine clinch the diagnosis.

Management

General measures

Gastrointestinal decontamination with active charcoal is indicated. Massive overdose requires respiratory support.

Specific measures

Flumazenil is a competitive benzodiazepine receptor antagonist that reverses the CNS and respiratory depression. As it is a short acting drug, it should be given in incremental doses of 0.2, 0.3. and 0.4 mg at 1 minute intervals intravenously. Failure to respond to this therapy rules out benzodiazepine poisoning. (Prischl et al., 1988).

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